

12700
17
Omnibus

ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY.

VOL. XXIII.

DECEMBER, 1914.

No. 4.

LV.

FURTHER OBSERVATION ON SOME ANATOMIC
AND CLINICAL RELATIONS OF THE SPHENOID
SINUS TO THE CAVERNOUS SINUS AND
THE THIRD, FOURTH, FIFTH, SIXTH
AND VIDIAN NERVES.*

BY GREENFIELD SLUDER, M. D.,

ST. LOUIS.

Last May it was my privilege to present this subject to this association in the shape of a preliminary report. Now I should like to add a note to it. The subject is a large one and clinically deals with a most difficult class of cases. It may be recalled that the question of the etiology and treatment of migraine was raised in that report; and the statement made that I believed many (but not all) of the recurrent headaches that bear the name migraine are sphenoidal empyemata that had lost most if not all local signs; or were started as such empyemata; and that the nerve trunks had become involved either by extension of the inflammation (or its toxin) through the thin wall separating the sphenoid sinus from the adjacent

*Read before the American Laryngological Association, May 5, 1913.

nerve trunks. The results (case reports) that I might now submit, although a goodly number, are too recent to permit of conclusions. I feel that these results should stand five years before conclusions are drawn. As far as they go, however, they strengthen my belief in the correctness of the deductions detailed in that report.

I desire now specially to record an anatomic observation and to mention a conclusion drawn from clinical observation as far as it has gone.

Last May I cited that the treatises on anatomy usually gave the impression that the trunks of the third, fourth, fifth, sixth and Vidian nerves were well (far) separated from the sphenoid sinus. During the year an additional number of volumes have been consulted, but no exception to this rule has been found. The observation that the third, fourth and the three divisions of the fifth, sixth and Vidian frequently lie in close association to the sphenoid sinus was a deduction from specimens which I had studied by cross sections. Two months later (July 18, 1912), Ladislaus Onodi,¹ reported an anatomic investigation of this same district, with corresponding findings, save for the Vidian nerve, which he did not observe. His method was to follow the nerve trunks in certain specimens, sometimes to remove the wall of the sphenoidal sinus, and then to study the relations of the nerve trunks thereby exposed. He found that they were in these close associations for varying distances, sometimes even as much as twenty millimeters. He did not consider the cavernous sinus in these relationships. He pictured specimens, however, where the sphenoid sinus extended close to the clivus of Blumenbach—so close as to make transparency of the separating bone—and showed how this brings the sixth nerve into these associations. I have not such a specimen. Five years ago, while looking over the exhibit of Dr. Mosher in the Harvard Medical School, I saw a number of such; but none of sufficiently thin wall to suggest their being classed with such material as I drew my conclusions from. It is interesting to know the length of such contacts; but I believe this to be of secondary importance to the fact that the exposure or contact exists, however short or long. Last February I enjoyed the personal favor of an inspection with Dr. Warren B. Davis, Keen Research Fellow of the Jefferson Medical College, Phil-

adelphia, of his matchless collection of one hundred and forty-five Caucasian specimens, showing the nose and accessory sinuses from the eighth week of fetal life to the twenty-fifth year uninterrupted, several specimens for each year (save the eleventh year, of which there is only one).^{*} With his sanction I now record the observation made then, that the sphenoid sinus spreads laterally at an early age, reaching to close proximity to the second division of the fifth as early as two and one-half years; and that this condition runs almost constant throughout the series. Its development (Davis) begins on the anterolateral aspect of the body and slowly extends backward. It, however, spreads rapidly laterally, to approach the foramen rotundum, and then proceeds backward. As early as the sixth year the Vidian canal may be approached.

These observations seem to me of great importance. They furnish anatomic findings to correspond with the clinical histories of this class of cases, i. e., that the neuralgias frequently begin quite early in life. For if I be right in the conclusion that the mode of production of these headaches—the pathologic sequence—is the close association of the sphenoid sinus to these nerve trunks, and that the inflammatory processes are transmitted through the thin bone separating the cavity of the sphenoid from the associated nerve trunks, it is necessary that such anatomic associations be formed in early life, as an explanation of such headaches beginning in early life.

Last May I made the statement that I thought the pathologic process underlying these cases to be a hyperplastic sphenoiditis. A year more of clinical observation strengthens this belief, although some cases are certainly not such, according to their clinical behavior, and bone removed from some of these patients was declared to be normal by Dr. E. L. Opie.

From an observation of about one hundred cases it has seemed that the second division of the fifth and the Vidian are the nerves most frequently involved (ninety-five per cent). They may be involved singly or together, then making the picture which would otherwise emanate from the sphenoid.

^{*}These specimens were secured by the ingenious technic which Dr. Davis was clever enough to devise, and which he will publish in his monograph giving his observations on the collection.

palatine ganglion. The year's observation leads me to feel it to be more difficult than I once thought to separate this class of cases from sphenopalatine ganglion neuralgia,² so that I feel that one ought to be carefully on guard for this differentiation.

The third nerve is rather frequently involved, but I cannot now give percentages. Such patients are usually unconscious of a difference in their pupils, but upon observation show this difference with almost every coryza. Observations relative to the fourth nerve are not so readily made. So far I have not seen a case of sixth nerve involvement belonging in this category.

During the year I have tried for intrasphenoidal use three medicines not mentioned in the first text. Iodid of potassium in water (two per cent to five per cent) proved to possess nothing upon which its use may be recommended. It is apparently inert. One per cent chlorotone in water proved to lack recommendations. The proprietary "cresatin" also failed to prove better adapted for these purposes than carbolic acid or oil of wintergreen.

The medicines which have so far given the best satisfaction are one per cent carbolic acid in oil, two per cent to ten per cent oil of wintergreen, and aqueous solutions of sodium salicylate, two to five per cent. These have been successful in allaying the pain long after the sinus was satisfactorily opened and the wound healed.

REFERENCES.

1. Onodi, Ladislaus: *Archiv. für Laryngologie*, B. XXVI, Heft 2, July, 1912.
2. Sluder, Greenfield: *The Syndrome of Sphenopalatine Ganglion Neurosis*. *American Journal of Medical Sciences*, December, 1910.

LVI.

THE VALUE OF EYE SYMPTOMS IN THE DIAGNOSIS OF OBSCURE CHRONIC SINUS DISEASE.*

BY LUTHER C. PETER, A. M., M. D.,

PHILADELPHIA.

There is some difference in opinion as to the frequency of eye symptoms in sinus disease. This is due to several causes. First, the average sinus case is not referred to the ophthalmologist for careful study unless eye symptoms are so pronounced that the patient is conscious of damage to his sight.

Second, ophthalmoscopic examinations in retrobulbar conditions are frequently negative, and a careful study of the fields for form and color is often omitted when central vision is good and the eye ground is negative.

Third, the character of the sinus disease, as to whether it is acute or chronic, will influence the observations as to the relative frequency of eye complications.

The diagnosis of acute sinus disease, as a rule, is more readily made than the more serious chronic form—the latter being more serious because of its chronicity and the resulting irreparable damage to the sinuses and the structures surrounding them. Eye symptoms are not infrequent complications in the acute period, but diagnosis, as a rule, is made and treatment instituted before serious damage is done to the eye structures. This possibly gives rise to the erroneous impression that eye symptoms are not common in sinusitis. It is, however, in the chronic variety that we meet with the really grave complications, and among these complications the eye is most frequently involved. A casual glance at the human skull, with almost parchment-like walls separating the orbit from the sinuses, and a knowledge of the blood vessels and drainage system of the nose and orbit, should be convincing

*Read before the North Branch of the Philadelphia County Medical Society.

evidence that chronic disease of the sinuses must, in time, extend to the orbital structures, if the progress of the disease is not stayed. This, therefore, should serve as a warning to be on the lookout for eye symptoms in chronic sinusitis.

From a clinical standpoint we have abundant evidence of the coexistence of disease in these structures, and in many instances the eye furnishes the clue to the situation. I do not intend to give you a classified list of changes which we find in eye complications, but wish to call attention to a few which will not only help the rhinologist, but the general practitioner as well, to arrive at definite conclusions in some of these puzzling cases.

The avenues of extension of disease of the sinus to the orbit are practically two—either by contiguity, or through the lymph and blood vessels. So close are the posterior ethmoid and sphenoid walls to the orbit that necrosis of the walls of these sinuses means necrosis of the orbit. The dural sheath of the optic nerve is continuous with the periosteum of the orbit as the nerve passes through the orbital canal. The optic nerve, therefore, is unprotected, and the nerve is open to the slightest inflammation. Strange as it may seem, the periphery of the nerve, and the part directly in contact with the point of inflammation, does not always show the evidence of disease to the same extent as the central bundle, known as the papillomacular bundle, of nerve fibers. This highly specialized bundle which carries impressions to and from the macula and brain, and composes about one-third of the diameter of the nerve, is centrally located in this region. Possibly the resulting scotoma or blind spot is one of the earliest evidences of optic nerve involvement in chronic sinus disease. It is erroneously assumed by some that this papillomacular bundle in the orbital canal comes very close to the surface, and for this reason promptly suffers in chronic sinus troubles. A short distance back of the globe, it does approach very close to the surface; but at the part of the nerve which probably is the seat of disease, shortly after it passes through the optic foramen, this bundle occupies the center of the optic nerve. It is at this point that damage to the nerve first appears. It is also interesting to note that in the retrobulbar neuritis due to toxemia of sinus disease, as in other forms of toxemic retrobulbar neuritis, this papillomacular bundle of nerves is

usually first involved. This is due to the fact, possibly, that these nerve fibers are more highly specialized, and therefore more delicate in structure, than the rest of the optic nerve.

Cases in which eye symptoms are of most value, as a rule, consult the ophthalmologist first, and are referred by him to the rhinologist for surgical intervention. The usual symptom is a rapid lowering of vision in one eye. In my work I have learned to look upon a rapid unilateral lowering of vision, without apparent cause, as pathognomonic of chronic disease of one or more of the sinuses—usually, the posterior ethmoid and sphenoid—and occasionally antrum disease. The frontal sinuses rarely give rise to this condition.

The following cases confirm this:

F. F., aged seventeen years. Admitted to hospital September 5, 1913. Gradually lost vision in the right eye during ten days previous to admission; can now only see hand movements at one foot; has no pain, no discharge from the nose. Eye grounds are negative, except for slight venous engorgement in the right eye and some striation of the retina. A tentative diagnosis of probable sinus disease was made. He was placed under pilocarpin sweats daily, and was referred to the rhinologist for examination. The first report from the nose and throat department was "probably negative." An X-ray examination and removal of the middle turbinate on the right side were requested. The X-ray showed a shadow in the right antrum. The middle turbinate was removed on the right side, and the posterior ethmoid and sphenoid cells were found to be necrotic and contained pus. Curettage and drainage were followed immediately by rapid rise in vision, and now central vision is 20/20.

The case illustrates the rapidity of the onset of the diffuse retrobulbar neuritis—requiring five days to be so marked as to reduce sight to hand movements at one foot. He further illustrates the dangers of such a condition. Notwithstanding prompt relief, he now has a secondary atrophy of the optic nerve of moderate degree. A fact worthy of noting is this—that improvement in the eye condition remained stationary four or five days after opening up of the ethmoid and sphenoid, and improvement was again observed, and remained permanent, after tapping and irrigating the antrum on the right side, which also contained pus.

The second case illustrates a slightly different phase of eye complication. His history is, briefly, as follows:

Mr. L., aged twenty-three years, called at the hospital on the 22d of September, complaining of lowered vision in the right eye. A month previous to this he had four polyps removed from the right side of the nose. Twenty-four hours after the operation the face was swollen, the lids closed, and in a short time sight was reduced to hand movements at one foot. Pus accumulated in the orbital cavity and was released by an incision in the lower lid. Recovery was prompt and vision improved. He consulted us because his vision again became lowered. Careful examination of the nose was made, and five large polyps were again removed from the right side of the nose. The ethmoid and sphenoid cells were curetted and irrigated, with the immediate improvement of vision from 20/70 to 20/30.

It is interesting to note in this case, also, that improvement was not complete until the antrum on the same side had been trephined and irrigated and relieved of its pus contents. This patient has clearly pointed out a symptom which is common among these chronic sinus diseases—namely, a central scotoma. The patient complains of a white object, especially on looking towards the right. When his field was carefully mapped out, we found that he had a pericentral scotoma—the more central of the macular fibers having escaped. This, therefore, is an illustration of disease of the optic nerve by pressure upon the sinus walls—the enlarged polypoid growth having closed up the ethmoid and sphenoid openings, thereby causing retention of pus. The patient now has a mild form of optic atrophy, following the retrobulbar neuritis.

Neuroretinitis is more common in acute forms, from direct pressure, and when present is usually recognized because the patient complains of lowered vision. The fact, however, that the patient does not complain of visual disturbance, does not guarantee an intact visual field. If sinus diseases were routinely referred to the ophthalmologist for joint study, many early evidences of eye complications might be detected, and permanent damage to the visual tracts might be avoided. We are apt to forget that scar tissue in the nose may not be serious, and may not cause inconvenience to the patient—but

scar tissue in the optic nerve and retina means irreparable damage.

A word as to the enlargement of the normal blind spot. I have never been able to observe an enlargement of this normal blind spot, except in patients in whom papillitis or neuroretinitis was present. What may be mistaken for an enlarged blind spot is a central scotoma, in which the more central fibers of the macula have escaped damage. This is not unusual, and the second case reported is illustrative of this form of scotoma.

Unilateral loss of accommodation and disturbance of muscle movements and muscle balance are occasionally observed. Either symptom, however, is usually accompanied by other ocular manifestations which are more pronounced. I have referred in the beginning of my paper to the fact that many of these cases with visual complications may show no change whatever in the ophthalmoscopic picture. The importance, therefore, of careful studies with the perimeter and scotometer must be apparent at least to those who do eye work. The fact that the peripheral retinal layers contain relatively few rods and cones, that they are insensitive and record relatively few impressions, often prevents a hurried and perfunctory study on the perimeter from showing any marked peripheral change. More careful study of this peripheral retinal area sometimes reveals narrowing and cutting in the fields quite as early as the changes which are characteristic in the macular region.

From this hurried discussion of some of the eye complications of chronic accessory sinus disease, you will observe that they practically fall into two groups: First, gross changes, or those which may be observed, at least in their effects on the optic structures, by one who is not especially skilled in ophthalmic examinations; second, those changes which can only be determined by most careful technical examination.

Of the value of the former group of changes there can be no question. When present they are part of the symptom complex of chronic disease of the sphenoid, posterior ethmoid and antrum. The latter group is of even greater value, because they serve warning of approaching damage to the optic nerve, and, as a rule, this warning is served in time to avoid serious permanent injury.

Conclusions which force themselves upon us, from a study of these cases, are: First, that chronic disease of the accessory sinuses should be studied not only from a rhinologic, but from an ophthalmologic standpoint as well; second, such studies should be made early, even though no gross optic nerve involvement is manifest; third, when gross evidence of disease of the optic nerve is present there should be no hesitancy in promptly sacrificing the middle turbinate to thoroughly open up the sphenoid and ethmoid cells; fourth, all accessory sinuses should be explored to determine their condition when eye complications appear.

LVII.

LEPROSY OF THE UPPER RESPIRATORY TRACT,
WITH REPORT OF A CASE.

BY JOHN HORN, M. D.,

NEW YORK.

Cases of leprosy, not being endemic, are comparatively rare in this country. Up to the present time relatively few cases have been observed in the United States, and such as have been seen were of the mild type, which accounts for their undetected passing of the quarantine station. In 1902 the United States Leprosy Commission found two hundred and seventy-eight cases in this country. Of these individuals, one hundred and eighty-six probably contracted the disease in this country; one hundred and twenty were born in foreign countries, and one hundred and forty-five were native born. The Sandwich Islands became infected in the nineteenth century.

The contagiousness of leprosy appears to have been recognized at a very early period. In 636 A. D. leprosy houses were instituted in Italy and other countries, and the practice of segregating lepers soon became general. It is a prehistoric disease, having been first known in Egypt and the Orient. About 345 B. C. it was found in Greece, and in about the first century B. C. in Italy, and later extended to other European countries. In Great Britain it is known to have existed as early as the tenth century, and from this country it was carried to Iceland and Greenland. South America and the West Indies were most likely infected from Spain. Its causative factor, a small bacillus, was first described by Hansen in 1870, who found it in the tissues, both extra- and intracellular. In its morphologic and tinctorial qualities the leprosy bacillus resembles the tubercle bacillus. Experiment animals seem to be nonsusceptible to infection, and up to now there has been no success in growing the bacillus on artificial media, though Arning claims to have transmitted the disease to a rabbit.

It is thought that the disease is chiefly disseminated by the secretions, mostly from the nose and upper respiratory tract, as well as from the surfaces of leprous ulcers, though the bacilli from the secretions seem to be of a degenerate form, and this may lessen their infectiousness in these substances and account for their low character in this respect. Intimate and prolonged association with the diseased and uncleanness play an important rôle in its transmission; the wife of a leprous husband eventually becomes infected, and the children of lepers often develop the disease in early life. Indirect contact may have something to do with its transmission, as witnessed by the number of lepers among the laundresses in an infected locality. Almost constant and early appearing lesions in the nasal passages led Stricker to believe that the latter constituted the chief infection atrium. Hereditary acquisition is now doubtful.

A characteristic of the disease is the presence of large masses of bacilli in the leprous tissue; they are mostly intracellular and in groups, and are probably carried to distant parts of the body through the lymphatics. Characteristic also of leprous tissue are the large vacuolated cells, the lepra cells of Virchow, and the globi of Hansen, which are filled to bursting with the leprosy bacilli. These bodies were considered by Unna and others as zoöglar masses rather than intracellular accumulations, and Kanthak interprets them as bacillary thrombi in the lymphatic vessels. The nodules or lepromas consist of granulation tissue containing many round and epithelioid cells, lepra cells, and occasionally multinuclear giant cells. While the bacilli are most numerous in the nodules, they are also found in any organ or tissue invaded by the disease, such as the liver, spleen, kidney, the nerves, blood vessels, etc. It is thought that the bacillus secretes little or no soluble toxin. Failure to cultivate the bacillus has prevented the preparation of a suitable serum. It seems probable that all men are susceptible to leprosy under proper conditions. Santon states that children from four to five years are particularly liable to infection. (In the above description liberal use has been made of Rickett's¹ work.)

Many of us are only familiar with leprosy from paper illustrations, though the writer was fortunate enough to have a leprous patient referred to him on account of severe hoarseness

and because the larynx showed a definite picture of leprosy involvement. This case has up till now only been presented to dermatologists. To the laryngologist, of course, the cutaneous lesions are only of secondary interest, and only the condition of the upper respiratory tract and the laryngeal lesions in particular will be considered here. Before describing the details of the larynx and the observations made of the leprosy patient by the writer, it may be opportune to briefly abstract from the lepra literature of the past concerning the invasion by leprosy of the upper respiratory tract and larynx; among the chief observers were Arning,² Schmidt,³ Gerber,⁴ Manges,⁵ Hollmann,⁶ Fox,⁷ Kerl,⁸ Bergengrün,⁹ and Babes,¹⁰ who all agree that leprosy shows a predilection for affecting the upper respiratory tract. Otherwise the leprosy infiltration seems to advance from the cutaneous surface to the nasal cavities, often limiting itself to the anterior portion rather than to the post-nasal and nasopharyngeal cavities; yet that the nasopharyngeal cavity is affected was shown by Arning's study of the lepra cases on the Sandwich Islands, where a great many of these cases have shown the leprosy infiltration involving the oropharynx at various stages of development.

Schmidt examined a case of laryngeal leprosy and found the epiglottis thickened by a cauliflower excrescence, whitish in appearance, the aryepiglottic folds also thickened and nodular, and a small nodule was seen on the right false cord; in this case also the uvula was thickened, granular and covered by fine cross ridges; the hard palate also showed a bluish nodule. In these cases the leprosy nodules of the mucous membrane are soon cause for complaint, and as they grow larger by increasing infiltration and more numerous, narrowing the lumen of the nasal cavities, pharynx and larynx, an element of danger is added to the discomfort of the leprosy patient. More severe than the nasal and pharyngeal distress is of course to be expected from the laryngeal stenosis.

Bergengrün differentiates three stages of mucous membrane lepra: (1) The stage of catarrh and erythema, (2) the stage of infiltration and tumor formation, (3) the stage of tumor necrosis with subsequent scar formation.

Laryngitis lepra begins at the epiglottis, the vocal cords becoming affected much later. The clinical symptoms are portrayed as beginning with an irritable cough, produced by a

ticklish sensation in the throat, being soon followed, however, by harassing cough with expectoration, to which is soon added hoarseness and eventually aphonia. The swelling and infiltration of the larynx by tumor masses, causes narrowing of the glottis and dyspnea; during the necrotic stage small particles of necrosed tumor or particles of food may close this small aperture and be the cause of choking spells. Babes, coincidently with Kalindero, was the first to describe the tumor formation of the larynx in leprosy. He claims the larynx is affected quite early in the disease, erosions and fine granulations being observed on the epiglottis, the disease process then extending to the arytenoids and vocal cords. In all cases the increased infiltration of the affected tissue assumes a remarkable coarse form of change, appearing as yellowish red, sometimes bluish, nodules, having often a glistening white center. In the oropharynx the tonsillar pillars, tonsils, post-pharyngeal walls, and the hard and soft palate may be infiltrated with leprous nodes; in the larynx, the epiglottis, aryepiglottic folds, arytenoid cartilages, false and true vocal cords, and the subcordal and tracheal mucous membrane participate in the pathologic process. Coarse ridges are oftentimes formed by the coalescence of several nodules. Disintegration of these leprous nodules into necrotic masses often entirely destroy by this process the bony and cartilaginous framework of the upper respiratory passages. Different stages of nodular development in this region are observed in close proximity in the one individual, large and small nodules uniting to form ridges, tumor formation and consecutively perichondritis, necrosis and edema.

The possibility of mistaking leprosy of the larynx for a tubercular or syphilitic process is not so remote, particularly when the leprous process presents an ulcerated form. In differential diagnosis it is important to keep in mind the fact to which all observers assent, that lepra of the upper respiratory tract is always a secondary disease process, whereas tuberculosis and lues of the larynx and nasopharynx are not at all seldom of primary origin. All leprous cases sooner or later show laryngeal involvement. In doubtful cases for differentiation recourse must be had to bacteriologic and histologic examinations, or else to inoculation of laboratory animals, which may prove of some worth; as noted before, Arning

claims to have been successful experimentally in causing lepra in a rabbit by inserting subcutaneously into the animal pieces of human leprosy tissue; he also successfully inoculated with leprosy a healthy condemned criminal shortly before his execution on the Sandwich Islands.

Laryngeal stenosis is the threatening danger in this insidious disease of the upper respiratory tract, and to relieve this condition tracheotomy is indicated. For leprosy as an infectious disease in general we know of no curative remedy, and the treatment can be only symptomatic and palliative, relieving the neuralgic and paresthetic phenomena as they arise by narcotic drugs, and attempting to improve the general condition with tonics and stimulants. Cauterization and curettement may temporarily improve, but have no permanent healing tendencies. The disease may extend over several decades, but often the patient succumbs at an early stage of the disease from exhaustion.

CASE REPORT.

Summarizing the family and clinical history, condition of the larynx on laryngologic examination, the therapy and subsequent course of the disease, the report is as follows:

L. T., female, single, aged twenty-seven years, native of Kurland, German Russia, came to the United States nine years ago. Her mother died of leprosy five years ago; the other members of the family have so far remained exempt from the disease. Other than an hereditary acquisition, no direct form of infection can be elicited from the patient. Before coming to the United States the patient was under special treatment for some nasal affection, which at that time, no doubt, was an unrecognized leprosy infection of the nose, and this was likewise overlooked by our quarantine officers upon her entrance into this country. Six years ago the patient came to Dr. Ludwig Oulmann, of New York, for dermatologic treatment, and from the clinical symptoms as well as from bacteriologic examination the diagnosis of leprosy was made. The bacillus of Hansen was at this time easily demonstrated in the blood as well as in the tissues. The cutaneous lesions being of less interest to the laryngologist, they are here omitted.

About three years ago the patient underwent some nasal operative treatment, with but slight relief to the local condi-

tion, and the treatment for the skin lesions by Dr. Oulmann exercised no permanent benefit. It may be mentioned that nothing was left undone that might have stayed the advance of the disease process, and along with other remedies "nastin," a leptothrix serum, and "salvarsan" were used hypodermatically without any favorable influence. The Wassermann reaction in this case was always strongly positive. Soon a persistent hoarseness developed, and the suspicion that the lepra process had invaded the vocal organs could be corroborated on laryngologic examination. This was two years ago, when the patient first came under my observation; the clinical picture then consisted of crust formations in the nose, with a large perforation of the nasal septum. Numerous nodules covered the remaining septal surfaces as well as the posterior pharyngeal wall. About the border of the epiglottis and arytenoid cartilages there are numerous coalescent nodules as well as some isolated, grayish pearly and glistening in appearance, with a yellowish brown coating. The false cords, especially on the left, are covered with nodules, and both are infiltrated. The left vocal cord is retracted and in a fixed position, otherwise the vocal cords seem to be free from infiltration. The patient has absolute aphonia and is barely able to whisper, has slight dysphagia, but nowhere are ulcerations to be seen.

As regards the therapy in this case, my report is unfavorable. The usual cauterization treatment and fulguration under cocain anesthesia yielded no apparent result after a fair trial.

In conclusion, it may be pointed out that the form of leprosy from which this patient suffers is not at all contagious, and hence the carrying out of any particular rules for isolation or quarantine is not thought to be necessary.

LITERATURE.

1. Rickett: Infection, Immunity and Serum Therapy, p. 455.
2. Arning: Berliner klin. Wochenschrift, 1890, p. 159.
3. Schmidt: Lehrbuch der Laryngologie, p. 350.
4. Gerber: Münchener med. Wochenschrift, 1910, No. 37, und Archiv. für Laryngologie, 1902, p. 126.
5. Manges: Zeitschrift für Laryngologie, Bd. III, 1910, p. 60.
6. Hollmann: New York Medical Journal, October 26, 1907.
7. Fox: Laryngoscope, April, 1908.
8. Kerl: Wiener klin. Wochenschrift, No. 4, 1911.
9. Bergengrün: Handbuch des Lar. und Rhin., Wien, 1898.
10. Babes: Spez. Path. und Therapie, Wien, 1901.

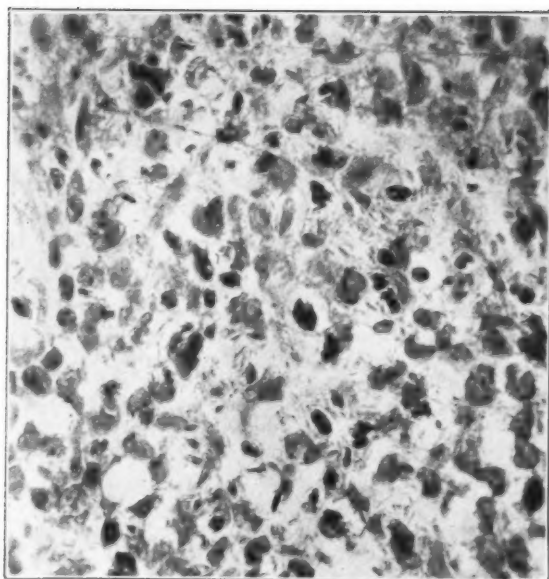


FIGURE I.

Microscopic specimen. Made by Dr. F. B. Humphreys.



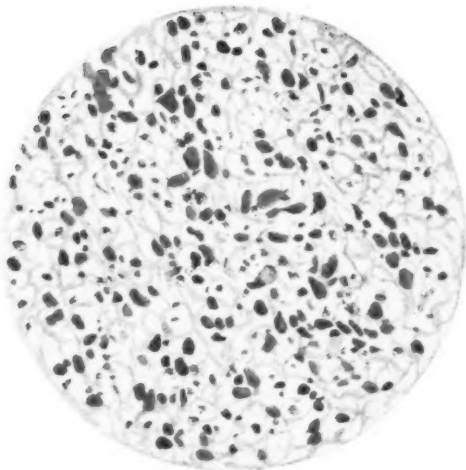


FIGURE II.

Microscopic specimen, showing Hansen's small rod-like leprosy bacilli colored red. Made by Dr. F. B. Humphreys.





FIGURE III.

Shows the condition of larynx
at time of writing.

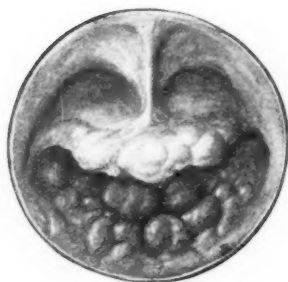
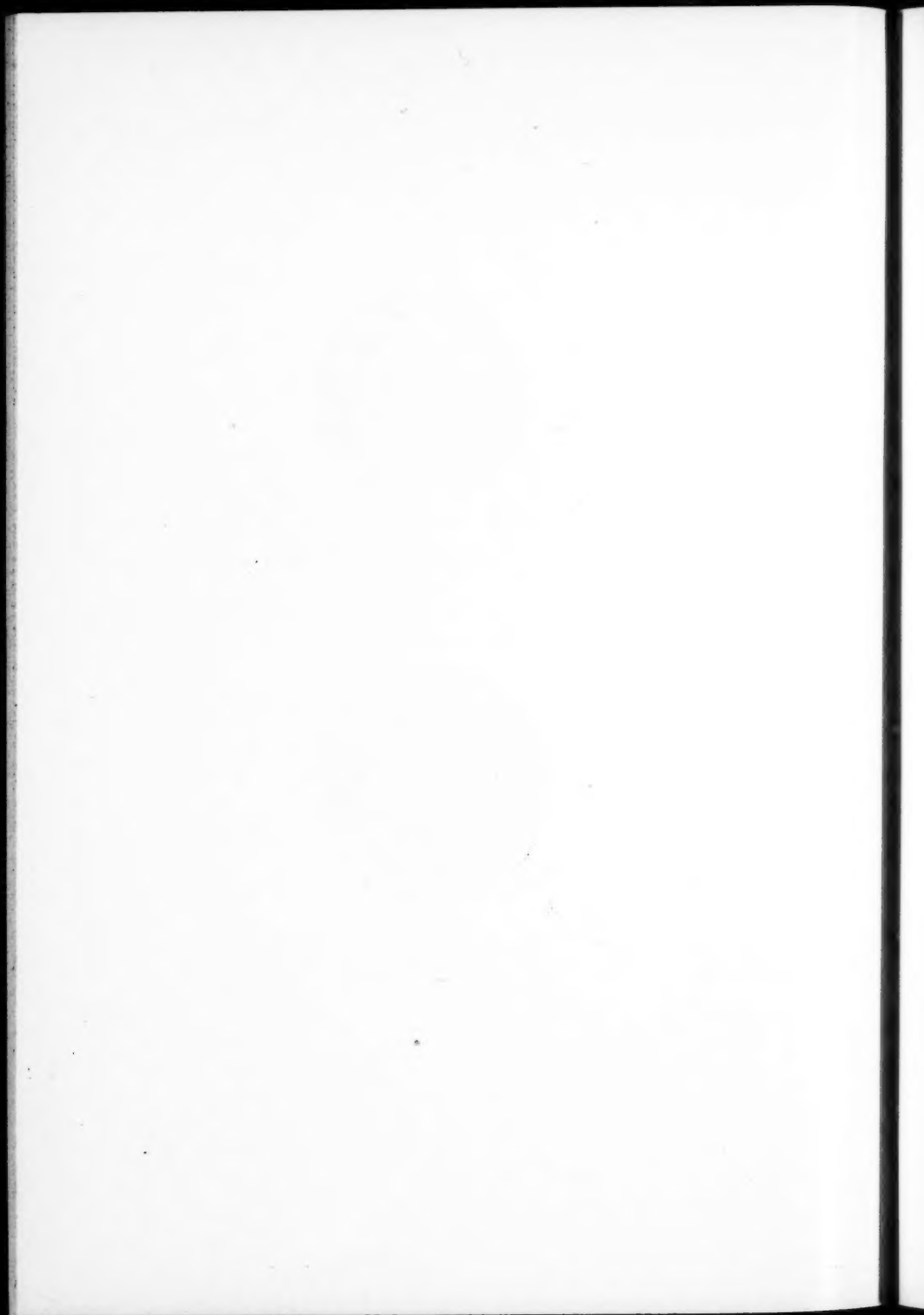


FIGURE IV.

Shows the condition of the
laryngeal region about six
months after.



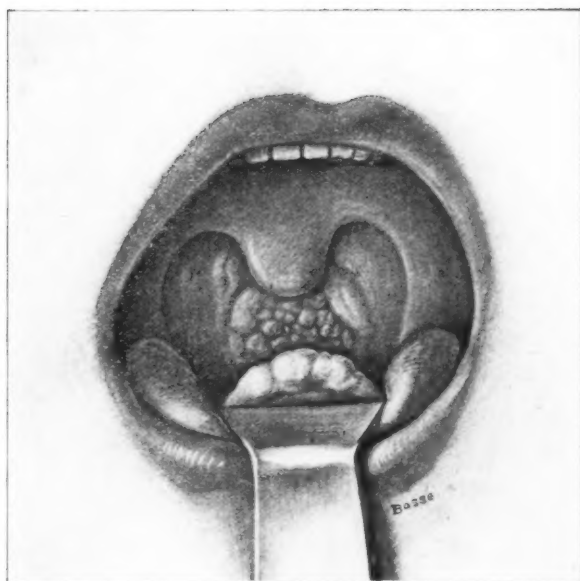


FIGURE V.

Shows the pharynx and upper part of the epiglottis. Recently taken.



LVIII.

THE DYNAMICS OF NASAL DEVELOPMENT—ITS
BEARING ON RESECTION OF THE SEPTUM.

BY WILLIAM WESLEY CARTER, A. M., M. D.,

NEW YORK CITY.

The importance of the nasal septum as a factor in the development of the nose, and the part it plays subsequently as an important integral part of the nasal arch, has been impressed upon me in the course of my work on the deformities of the nose. It is a subject, too, that has been largely neglected, though the importance of its bearing upon the submucous operation is recognized at once. That this function of the septum is real and of great practical importance to the surgeon has been demonstrated to me by the several cases of sunken deformity of the nose, due directly or indirectly to the submucous operation, that have been referred to me for correction during the past few years. Such results are to a large extent avoidable, and I believe that a thorough understanding of the forces employed by nature in the development of the flattened nose of the infant into the prominent, shapely organ of the adult, will contribute largely to the prevention of such deplorable accidents which reflect undeserved discredit upon one of the most artistic and useful operations in rhinology. In no operation do manual dexterity and experience count for more, and in no procedure do uniformly excellent results testify more eloquently to the skill of the surgeon.

When we consider the various forces that enter into the development of the nose, we cannot but marvel that the task imposed upon nature should ever result in the development of a symmetrical organ and one bearing a correct proportion to its facial environment.

Each side of the nose constitutes practically a dynamic entity, and yet these two systems must develop uniformly

and almost independently side by side to produce a single organ, upon the perfect, symmetrical development of which the comeliness of its possessor depends to a far greater extent than to any other factor.

Owing to the fact that the nose is a protuberance, springing from a more or less flat surface, and receiving its support, its impulse of growth and expansion from its base only, the poise of its constituent parts is all the more delicate and sensitive to disturbing influences.

The scope of this paper is not intended to include embryology, suffice it, therefore, to remind you that in the early fetus there is only one nasal cavity, and this does not exist as a separate chamber, but opens directly into the mouth. The division of this by the septum nasi commences before its separation from the mouth is effected by the palatal processes of the superior maxillæ and the horizontal plates of the palate bones. The fusion in the middle line of these three segments—i. e., the two palatal processes and the vomer—begins forward at about the eighth week and proceeds backward, and is completed about the tenth week. At this tripartite suture, if the union between the palatal processes is delayed or insecure, or if the lower edge of the vomer reaches this point of junction first, it may push down into the roof of the mouth and cause the ridge of bone frequently noticed in the midline of the hard palate. This ridge may also be caused later in life by the vertical pressure to which the septum is subjected during the formative period of the nose. The development of the flat infantile nose into the adult type is effected chiefly by forces acting from three separate and distinct sources—the two nasal processes of the superior maxillæ and the septum. To a disproportion in the action of these three forces is due the variations in the shape and contour of the nose which constitute so-called family and racial characteristics. To this may also be attributed the nontraumatic nasal deformities. The latter may be caused also by the unequal development of the two sides of the nose, especially the two primitive plates from which the vomer is developed.

Deflections of the septum at birth are unknown—in fact, they are almost never seen until the forces which take part in the development of the nose become active, which is between the fifth and seventh years. From this time on until puberty

there is every evidence of the activity of these forces. The most active period of all is during the second dentition, when the growth of the upper jaw is very active. At this time most of the nontraumatic deviations of the septum occur, for the septum is under considerable vertical tension, and the slightest force, such as the insertion of the finger into the nostril, may throw the septum out of the vertical, and being unable to recover its normal position, an obstructing deflection in time results.

Normal symmetrical development may be interfered with by many causes, but the most frequent is adenoids, which cause the upper jaw to be undersized and the arch of the palate to be raised. Under these circumstances the vertical space is too short to accommodate the normal increase in the length of the septum, and a deflection results.

The septum, which we will assume is the chief factor in the development of the nasal prominence, is locked in by the bony walls of the nasal cavity. Its vertical growth is opposed below by the arch of the hard palate, and above not only is it opposed, but it is being constantly encroached upon by the downward expansion of the base of the skull, due to the rapid growth of the child's brain; this influence is more pronounced in the highly civilized races, and corresponds exactly with our findings in regard to the frequency of septal deviations. These never occur in the lower animals, and they are extremely rare in the Mongolian and African races. Personally I have never seen a nontraumatic deviated septum in a negro, and the experience of several men with whom I have corresponded in regard to this matter, who have large practices in the South, corresponds with my own. In these races the nose is broad and flat, and the septum, in addition to being comparatively free from the encroachment of the brain box, does not seem to have, as in the Anglo-Saxon, the inherent power of growth necessary to raise the bridge of the nose. Certainly it is practically immune to those influences which, according to McKenzie and others, produce deviated septa in seventy-seven per cent of the Anglo-Saxon race.

The ossification of the various bones that enter into the formation of the nose begins at or about the same time—the sixth or seventh week of fetal life. The vomer, however, does not begin to ossify until the eighth week. This may

add somewhat to the disadvantage which its anatomic position imposes upon it, but the relative time at which ossification occurs in these bones does not aid us materially in our consideration of this subject, and we will turn, therefore, to the structure and position of some of the more important bones and to the general architectural construction of the nose.

In the opinion of the writer the septum has a most important function in the development of the nasal prominence. Its upper edge, where it lies between the lateral cartilages and between the nasal bones, constitutes the keystone of the nasal arch. But its chief function is that of raising the bridge of the nose, and this I believe is accomplished mainly by the vomer. I will, therefore, consider this bone more in detail. The vomer is placed like a wedge securely between the body of the sphenoid and the arch of the hard palate. Its anterior border forms an inclined plane, the apex of which reaches as far forward as the incisor crest. Its position is very secure, and it is well adapted to serve as a basis from which the force which develops the nose may spring. Furthermore, the anterior border of the vomer is parallel with the prospective contour of the nose. The growing vomer, therefore, acts at right angles upon the vertical plate of the ethmoid and the septal cartilage, and the growth of the latter in a downward and backward direction is well resisted by the vomer. The division of the original nasal cavity is effected by the vomerine cartilage; the anterior part of this remains as the septal cartilage, but the posterior part does not become the vomer. Ossification in the vomer does not occur in cartilage, but it begins in two ossific centers situated in the lower, back part of the membrane which covers the vomerine cartilage on either side. From these centers, which make their appearance about the end of the second month, ossification proceeds forward and upward. The fusion of these two lamellæ occurs behind and below about the third month, and their fusion gradually extends forward, absorbing and to a certain extent pushing forward the central cartilage and the vertical plate of the ethmoid. This process continues until puberty; at this time the laminae of the vomer have become completely united, and all that remains of the original cleft is the groove on its anterior surface in which is lodged the septal cartilage. The manner in which this cleft is closed,

beginning posteriorly and gradually extending forward, and this process continuing during the years of active nasal development, shows conclusively the importance of the vomer in the dynamics of the nose. On the other hand, if through disease such as syphilis or atrophic rhinitis the vomer is destroyed, or if its development is interfered with in early life, a saddle-back deformity, more or less pronounced, develops.

My studies have been conducted more with a view to solving the dynamics of the septum than of any other part of the nose, because of its practical bearing on the submucous operation and the influence which it has in the production of nasal deformities.

I have concluded, first, the normal position of the upper edge of the septum, which constitutes the keystone, is necessary in order to maintain the integrity of the nasal arch. In removing the septum, therefore, no instrument should be used that necessitates tugging, for if this upper edge of the septum is removed or displaced, a depressed deformity will result, which can be corrected only by the transplantation of bone. As I first suggested several years ago, a punch forceps is the best instrument for removing the septum. This not only requires no tugging, but it has the additional advantage of enabling one to remove the exact amount of tissue necessary to relieve the obstruction. I have concluded, further, that the lifting force exerted by the septum is indispensable to the development of a symmetrical nose, and therefore it cannot be extensively removed with safety during the years of active nasal development. Fourteen years of age I regard as the limit of safety. On the other hand, the framework of the nose, conforming as it does to the definition of the arch, does not require any external support other than at its two extremities. Therefore, the septum, if properly removed after the nose has acquired the correct proportions of adult life, causes no injury from an esthetic point of view, and the architectural strength of the nasal arch remains unimpaired.

LIX.

REPORT OF CASES OF AURAL INFECTION WITH
THE STREPTOCOCCUS CAPSULATUS.

BY CHARLES E. PERKINS, M. D.,

NEW YORK.

I desire to report briefly a few cases of aural infection with the streptococcus capsulatus, or bacillus mucosus, which illustrate some of the forms that infection with this germ may assume.

Case 1.—An instance of an apparently severe form of infection which cleared up by free drainage through the drum membrane.

M. E., aged thirty-three years, came under my care April 14, 1912. One week previously, while bathing in a pool, he was nearly drowned and was resuscitated with some difficulty. This was a common pool, about ten feet square, and one may presuppose the presence of germs in abundance. On the following day he had pain in the left ear, which became severe and was followed in a few days by pain behind the ear.

On examination he had marked tenderness; the drum membrane was bulging, thickened, and without luster. A free incision was made. During the next two days the discharge was profuse and the mastoid tenderness diminished, but pain in the opposite ear developed, with bulging membrane. This was freely incised under gas, and as the first ear was not draining properly, it was reincised. Four days later, reincision of the right, or second ear involved, became necessary, as it was not draining satisfactorily—as shown by inspection of canal, increase of mastoid tenderness, and the presence of some spontaneous pain. After this the case progressed satisfactorily, and the patient was discharged on the eighth day. I saw him, however, at times during the following three

weeks, when recovery seemed complete. I have lately examined this patient, after two years, and he has remained well.

During the last three years there have been quite a number of patients with this infection in Dr. Dench's clinic at the New York Eye and Ear Infirmary that I have treated and that have cleared up without operation. In some no special mastoid symptoms have been present at any time. Simply being suspicious of the character of the discharge, I have had a smear examined and having found the prevailing germ to be the streptococcus capsulatus, I have kept them under close observation and they have cleared up without mastoid symptoms. Some of them I have seen for two or three months, and am satisfied that their recovery was complete. I regret that I cannot give X-ray findings in these cases, but most of them occurred before we were accustomed to place as much reliance in X-ray plates as we do now. I have seen a number of cases of mastoid involvement which at no time were accompanied with aural discharge.

Case 2.—S. L., female, aged twenty-five years, referred by her physician on July 27, 1909. For three months had a small swelling behind the left ear, which appeared just subsequent to confinement. Examination showed a hard fluctuating mass behind and above the ear, about an inch in diameter. The auricle seemed to stand away from the head. The external auditory canal, the membrana tympani and fundus appeared absolutely normal. I advised postauricular exploration, and at the infirmary I operated on the following day and found the mastoid completely broken down, a large perisinus abscess, and a large quantity of pus. The tympanic vault was completely walled off by firmly organized tissue, which I did not disturb. Examination of the pus showed infection to be streptococcus capsulatus. The patient made a perfect recovery. At no time was there the slightest discharge from the canal. The wound was completely healed in five weeks.

Case 3.—Mrs. E., aged sixty-two years, consulted me on September 18, 1913. Began with pain in left ear, following a cold in preceding March, i. e., six months previously, during which time she had suffered more or less pain in the ear and head. She was treated by the family physician in all manner of ways except by incision of drum.

Examination showed some loss of luster and thickening of the membrana tympani, and also slight narrowing of the canal, with mastoid tenderness and thickening of postauricular tissues. Two and one-half per cent of sugar in urine; no acetone.

X-ray examination by Dr. Dixon: Large pneumatic type; septa between cells blurred. Frankly an operative case.

On account of the marked glycosuria, operation was done as rapidly as possible—total time, twenty-three minutes. The mastoid was found to be totally disorganized. Large perisinus abscess, with immense granulations springing from its walls; pus, abundant, showed infection to be streptococcus capsulatus. Wound healed in six weeks; remains well.

Case 4.—M. H., age sixty-five years; referred by his physician (on December 20, 1913), who had treated him for about four weeks for tinnitus and impaired hearing in the left ear. There was no pain or mastoid tenderness, but the drum was thickened and without luster. Inflation produced moist râles.

X-ray by Dr. Dixon: Right, clear pneumatic mastoid; left, same type, but cloudy throughout.

It was not until a week later that tenderness about one inch posterior to the ear developed and he began to have pain at night, then he consented to operation, which showed the mastoid to be disorganized and filled with a large granulating mass springing from the sinus. Large tip cells filled with pus, and an epidural abscess extending fully an inch posterior to the sinus in the cerebellar fossa. Convalescence uneventful; wound healed in five and a half weeks.

Case 5.—Mrs. M. S., age sixty-five years, consulted me on March 2, 1914, with a history of pain in the left ear for seven weeks. Four weeks previously, incision of membrane by a very competent man, followed by no discharge of pus. The mastoid was tender and the overlying tissues were somewhat edematous. The drum membrane was thickened and lusterless. There was no discharge from the canal. The operation showed the mastoid cells to be disorganized, with epidural abscess at sinus knee. Convalescence slow.

The following case is one of many observed in which the discharge was profuse, the bacteriologic examination leading, in a way, to the operation:

Case 6.—A. G., male, age fifty-eight years, came under my care February 12, 1914. Pain in right ear for two weeks. He was treated by his physician, but without incision of the drum. He had marked mastoid tenderness. The membrane, which was thickened, dull red, and lusterless, was incised. The following week the mastoid tenderness became very much less—indeed, nearly absent. The discharge, however, continued profuse.

X-ray by Dr. Dixon showed large celled pneumatic bone, cloudy throughout. Operation showed mastoid thoroughly infiltrated with pus. The wound healed in seven weeks.

The following case was one in which the ear infection seemed very benign:

Case 7.—Mr. S., age forty-five years, has had discharge from right ear for past two winters, lasting each time about one week. About the 1st of January, 1914, he developed an acute inflammation of the middle ear with spontaneous rupture of the membrane, which was incised two days later. The discharge was not profuse at any time. My impression is that I had a smear made and that it showed some benign infection, but I am unable to verify this, as the records are silent on this point. At any rate, the discharge had none of the characteristics which we are accustomed to associate with that due to streptococcus capsulatus. There developed marked spontaneous pain and tenderness in the occipital and preauricular regions; but, as there was no mastoid tenderness and the patient was a very nervous individual, this was attributed to neuralgia and hyperesthesia. Nevertheless, on January 16th, an X-ray was made by Dr. Dixon: "Left mastoid seems diploic; right (involved side), indistinct pneumatic; has appearance of sclerosed bone." One would hardly operate on this X-ray finding alone.

During February the discharge ceased, although inflation always showed fluid in the middle ear, and the membrana tympani remained dull and lusterless. Early in March a moderate sagging of the superior wall occurred, extending from Shrapnell's membrane outwards, about one-third the length of the canal. The pain and tenderness in the occipital and preauricular regions had become very slight, but a tender spot had developed about an inch posterior to the ear.

X-ray examination by Dr. Dixon showed (March 13th): Right mastoid, cloudy. Perisinus abscess has developed since January 16th (date of former examination).

(Note.—The beginning of the abscess was shown on former plate, but at that time was so small that it escaped notice.)

Operation on March 20th. Cortex, sclerosed, but after it was removed pus welled up and perisinus abscess was found. In this location—one inch posterior to ear—site of tender spot and abscess shown in X-ray plate, the bone was eroded nearly to the surface by a large granulation springing from the sinus. The patient made an uneventful recovery. The mastoid healed completely in four and a half weeks.

That one is not through with these cases at times, even when an operation has been performed and convalescence is apparently well established, is illustrated by the following case, occurring in the practice of Dr. N. R. Dann, to whose kindness I am indebted for the report:

Case 8.—W. K., age thirty-eight years, seen on December 4, 1913, in consultation with family physician. Aural sup-puration for three weeks. Examination showed profuse aural discharge, decided sagging of canal wall, and extreme mastoid tenderness. Operation same day. Two cortical perforations; pneumatic bone infiltrated with pus; sinus exposed. Wound practically healed on March 15th, over three months later, a small sinus, however, persisting. On March 25th, Dr. Dann saw the patient in consultation. He had suddenly gone into collapse. He had had severe headache for three days and nights previously, and had had severe vomiting. Temperature 102.4°. Patient unconscious and picking at the bed-clothes. Pupils unequal and do not react to light. Kernig marked. No lumbar puncture; no autopsy.

I think we are justified in concluding:

1. That this germ has a special affinity for bone tissue.
2. That in these aural infections, if the passage between the middle ear and the mastoid is large, drainage through the ear may effect a cure; so the importance of early and free incision of the drum membrane cannot be overestimated.
3. That the X-ray examination is of great help in these cases.

4. That an early and free operation is indicated as soon as we are convinced of irreparable mastoid involvement.

5. That these cases, whether operated or not, should be watched closely throughout, as late complications are more frequent, perhaps, than in other infections.

6. It would seem that patients advanced in life, and diabetics, are more liable to this infection than to other forms.

LX.

HISTOLOGIC PATHOLOGY OF THE NOSE.*

By JOSEPH C. BECK, M. D.,

CHICAGO.

In presenting this paper I wish to call attention to the paper I presented before this Section on "Histologic Pathology of the Accessory Sinuses" last year, which appeared in the Transactions, as well as in the ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY, December, 1913. The subject of this paper would be incomplete without considering the material already presented, since the accessory sinuses must be considered as part of the nose. However, in view of what has been published, I will confine myself to the pathology of the septum, inferior turbinated body, and organized exudates. The various changes that I have observed histologically are not always clearly demonstrable grossly, and, therefore, the importance of the microscope is essential in bringing out the pathologic changes in detail insofar as they may be applied to diagnosis, prognosis, course, and treatment.

I.—SEPTUM.

Figs. I to VII. From these sections one can very easily conclude that there is a definite change in the bone, in the presence of great rarefaction. The spaces are filled with the spongy material, not unlike that shown first by Siebenmann in the early stages of otosclerosis or spongification. The cartilage shows great activity at the junction of these spongifying changes in the bone. Similar changes have also been found in various other diseases, as acromegaly, osteomalacia, arthritis deformans, and physiologically in pregnancy in the first months. I have further demonstrated these same changes, early in the disease, in the middle turbinated body and ethmoid

*Read before the American Laryngological, Rhinological and Otological Association, Atlantic City, June, 1914.

cells in hyperplastic ethmoiditis and atrophic rhinitis. The later secondary changes are usually fibroses or scleroses in the bone and degenerative processes in the soft structures covering the bone. In my paper on "Histologic Pathology of the Accessory Sinuses," I advanced a theory, based on these pathologic changes, that the etiology of hyperplastic ethmoiditis and atrophic rhinitis, was possibly a disturbed function of the glands of internal secretion. This same theory or suggestion is again offered as to the cause of ridges and spurs in connection with the overgrowth of the septum and consequent deviation. Which gland or combination of glands is involved it is not possible to say at this time.

Cases of abscess of the septum which required subsequent resection to correct deformity show the loss of the cartilage cells with the formation of connective tissue, especially of the subperichondrial region and in the absorbed cartilage. (Fig. VIII.) The hyaline degeneration is seen in the homogeneous appearance. (Fig. IX.)

The bilateral thickening of the septal cartilage, otherwise spoken of as the tuberculum septi, is shown in Fig. X. There appears to be a great deal of activity in the cartilage cells, and the part just below the perichondrium, which the writer calls subperichondrium, appears very much thickened.

In connection with the filling out of a septum by means of paraffin in atrophic rhinitis the writer has chipped out a piece of cartilage with mucous membrane attached on one side, and in the several cases it shows, as in Fig. XI, that the epithelium is keratetic and thin, but the subepithelial structure and the perichondrium are thickened. The cartilage is unchanged. A statement made in textbooks as to the absence of blood vessels in cartilage appears not to be true, according to the specimen, shown in Figs. XII, XIII and XIV, in a case of cartilaginous deflection.

II.—INFERIOR TURBINATED BODY.

In regard to the inferior turbinated body we see the manifold changes in the soft tissues covering the bone. The latter (bone) is seldom changed. Occasionally in marked suppurative sinus disease there is an osteitis, but usually the changes are confined to the soft structures. These macroscopically

appear about the same, but histologically are quite different. The hypertrophy may be of the connective tissue, mucous glands, vascular, epithelial and bone. They may be and in fact are usually combined. The majority of specimens show the connective tissue predominating, and the glands and vessels are frequently destroyed by its pressure action. The epithelium is very much hypertrophied, especially in connection with chronic suppurative sinusitis. It is at times folded in and shows under the microscope like lakes or channels. (Figs. XVI and XVII.) Also some of these epithelial folds undergo hornification or degeneration. (Fig. XVIII.) The papillary changes of the posterior ends are principally made up of connective tissue covered with thick layers of epithelium. (Figs. XIX-XX.) These are spoken of as posterior hypertrophies or mulberry enlargements.

In the chronic intumescence of the inferior turbinate the connective tissue usually predominates; it is somewhat edematous and shows many blood vessels, especially of the newly formed type. The epithelium is frequently infiltrated with round cells. (Figs. XXI-XXII.)

In the study of atrophic rhinitis the writer removed a small particle of the inferior turbinated body and found that the change is not the same on both sides of the turbinate, as shown in Fig. XXIII. The mucous glands appear to lose their function by distention rather than by connective tissue contraction, as shown in both Figs. XXIII and XXIV.

I have observed that these various changes of the inferior turbinated body respond differently to various kinds of treatment, and that is the essential point in the study of these specimens. One therefore can prognosticate as to the recurrence after resection, crushing and cautery. The treatment that I most frequently employ, except in very large posterior ends, is the crushing of the lower edge by means of author's conchotribe (Fig. XXV), because it is applicable to the various pathologic changes mentioned above without destroying too much functioning tissue. I have made sections of these hypertrophies of the inferior turbinated body so crushed, and have found on the third day, at the height of the reaction (Fig. XXVI), marked round cell infiltration and areas of necrobiosis. Removing another piece on the tenth day, I found the tissue practically

all shrunken and very little evidence of organized tissue. (Fig. XXVII.) The result was correspondingly good as to breathing and drainage.

Syphilitic changes of the later stage are manifest as granulation, exudates and sequestra. The granulations and sequestra show microscopically the same picture as luetic granulation and sequestra anywhere else in the body, but the exudates lack the round cell infiltration found in luetic exudates elsewhere.

Sarcoma and carcinoma found in the inferior turbinated body are usually secondary to sarcoma of the nasopharynx and carcinoma of the superior maxilla (epulis).

The characteristic histologic findings of carcinoma and sarcoma are the same as anywhere else in the body.

Tuberculosis of the inferior turbinated body is very rare, and the one case the writer had and reported in Ballenger's textbook, page 304, showed the characteristics of giant cells.

True myxoma or polypi of inferior turbinate is also very rare, and the one specimen shown here (Fig. XXIX) is an evidence of the misinterpretation of the structure macroscopically. The inflammatory edema of the anterior or posterior end is usually mistaken for a true myxomatous polyp.

EXUDATES.

Almost every one has observed fibrinous casts after cautery, operations and infections. The histologic study of these are of considerable interest and importance, as shown in the illustrations. (Figs. XXX, XXXI, XXXII, XXXIII, and XXXIV.) I have been able to show that an exudate after electric cautery differs histologically as to the variety of the pathologic change in the inferior turbinate; i. e., the exudate is very much firmer and more fibrinous in the glandular hypertrophy than that of the connective tissue hypertrophy, and still greater than when the vascular tissue (turgescence) predominates. In this later condition there is frequently a recurrence of the exudate after it has disappeared, only not so marked. Macroscopically these exudates also differ in color and consistency.

The cause of exudates forming after operation is not known, but I have seen them in groups and have thought that it might be due to some chemical change in the cocain solution,

or to a mould formation within the solution. I have never found bacteria within them to any extent. During the time when I was experimenting with bismuth paste dressing after operation, I noted that fibrinous exudate infiltrated with bismuth were not at all uncommon.

The leucocyte infiltration is of particular note, often predominating over the round cells. I have found that in these cases where local applications, such as argyrol, were made, the exudate continued for a longer time than under other conditions. If it was removed mechanically it would reform very quickly.



FIGURE I.

Submucous resection of the septal ridge. Rarefaction of bone. Great activity of bone and cartilage at their junction.



FIGURE II.

Septal deviation. Ridge removed submucously, showing great cartilage and bone activity at their junction. Rarefaction clearly demonstrable.

Same as Figure I, higher power.



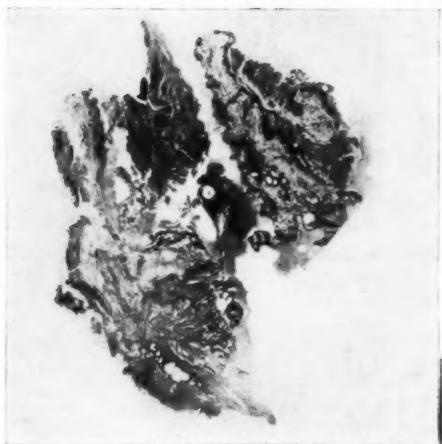


FIGURE III.

Ridge of the septum. Removed submucously. Showing marked rarefaction of the bone.



FIGURE IV.

Septal spur posterior portion near the sphenoid. Removed submucously. Showing great rarefaction in the bone with osteoclastic changes and interosseous substance filled with osteoblasts.



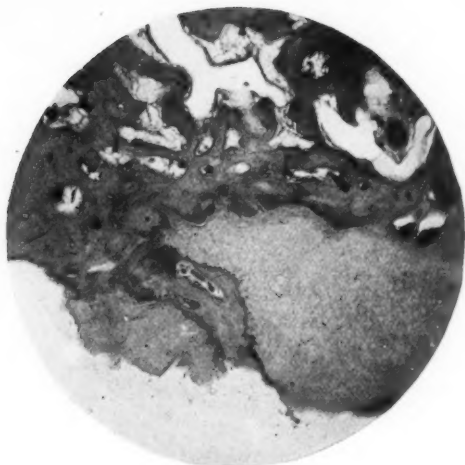


FIGURE V.

Ridge of bone removed submucously. Showing junction of cartilage and bone. Great activity at these points, with bony rarefaction.



FIGURE VI.

Large ridge from the premaxilla, showing marked rarefaction.

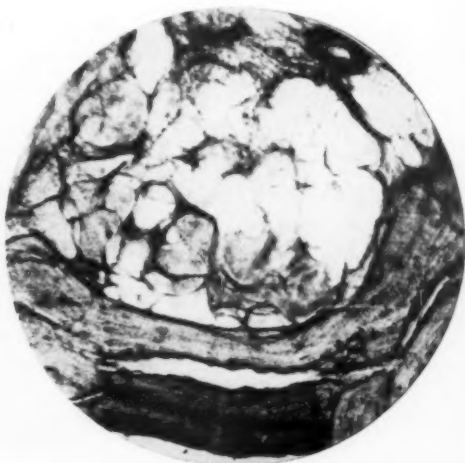


FIGURE VII.

Same specimen as Figure VI, higher power. Shows great deal of fat formation in the marrow.



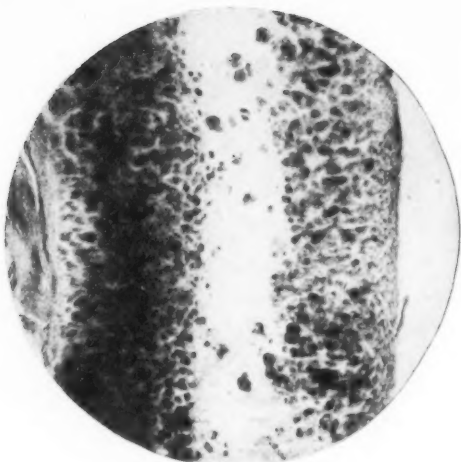


FIGURE VIII.

Abscess of the septum. Septum resected for deflection, following traumatic hematoma. Shows cartilage cells markedly infiltrated with round cells. Center of cartilage is homogeneous, and cartilage shows leucocytic infiltration.

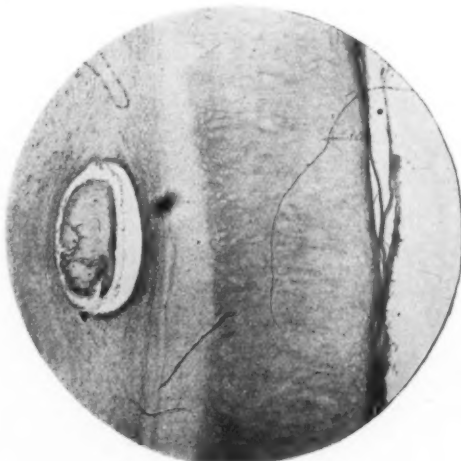


FIGURE IX.

Resected cartilage in a traumatic foot-ball nose. Caused primarily by abscess of the septum. Perichondrium very much thickened with large veins; organized blood within it. Cartilage cells in many places shriveled. Opposite side of perichondrium normal.



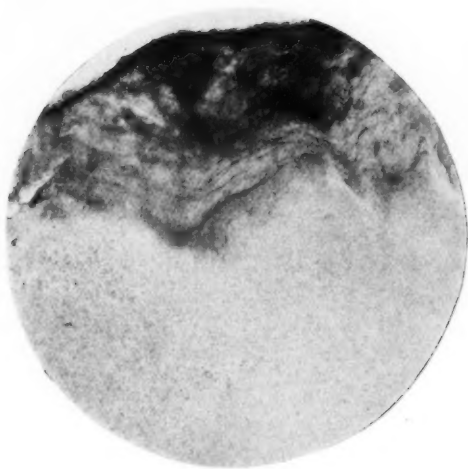


FIGURE X.

Deviation of the septum. Cartilage high up anteriorly. Young individual. Showing marked thickening of the subperichondrium with great karyoketic figures in the cartilaginous cells at that point.



FIGURE XI.

Sliver of septum in atrophic rhinitis (removed experimentally). Mucous membrane hyperplastic, especially the glands. Very thick perichondrium; epithelium thinner.



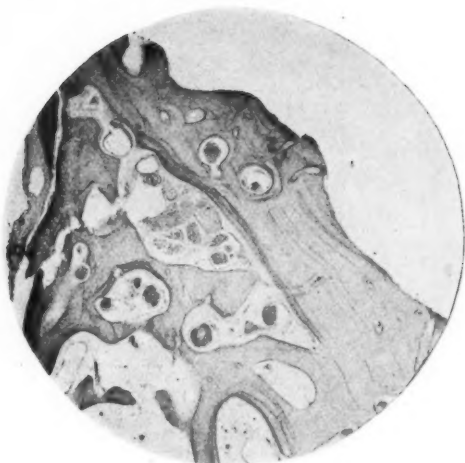


FIGURE XII.

Septal ridge showing at the junction of the bone and cartilage large blood vessel filled with blood. One of these vessels passes into the cartilage.



FIGURE XIII.

Same as Figure XII, higher power.



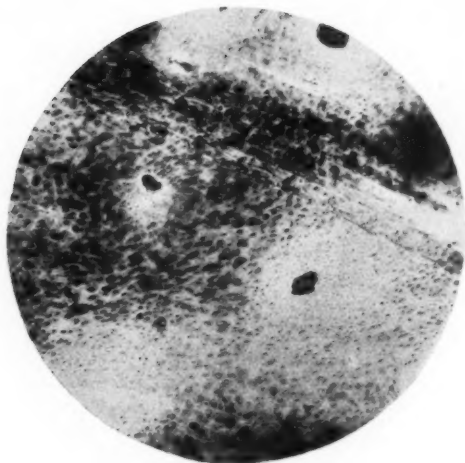


FIGURE XIV.

Septal cartilage showing a great round celled infiltration in the cartilage, and great activity of the cartilage cells themselves. Also cross-section of blood vessels.

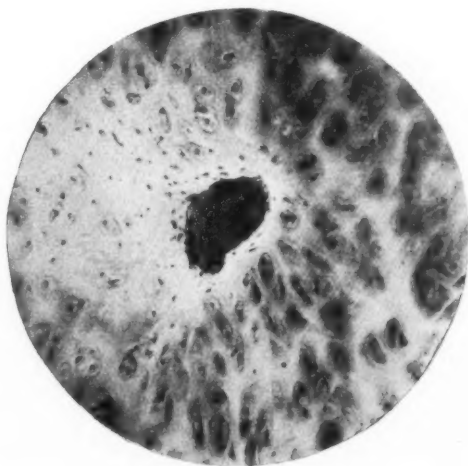


FIGURE XV.

Same as Figure XIV, higher power. Blood vessel filled with blood.





FIGURE XVI.

Hypertrophy of the inferior turbinate body. Mucous membrane showing great epithelial hypertrophy with folded-in masses.

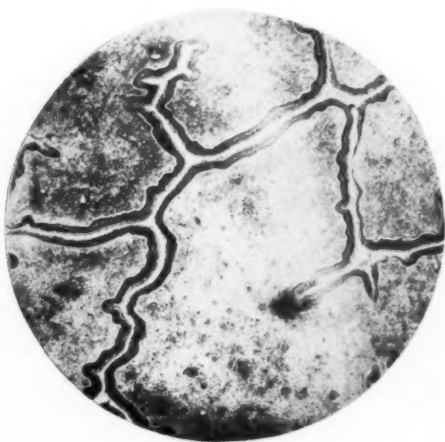
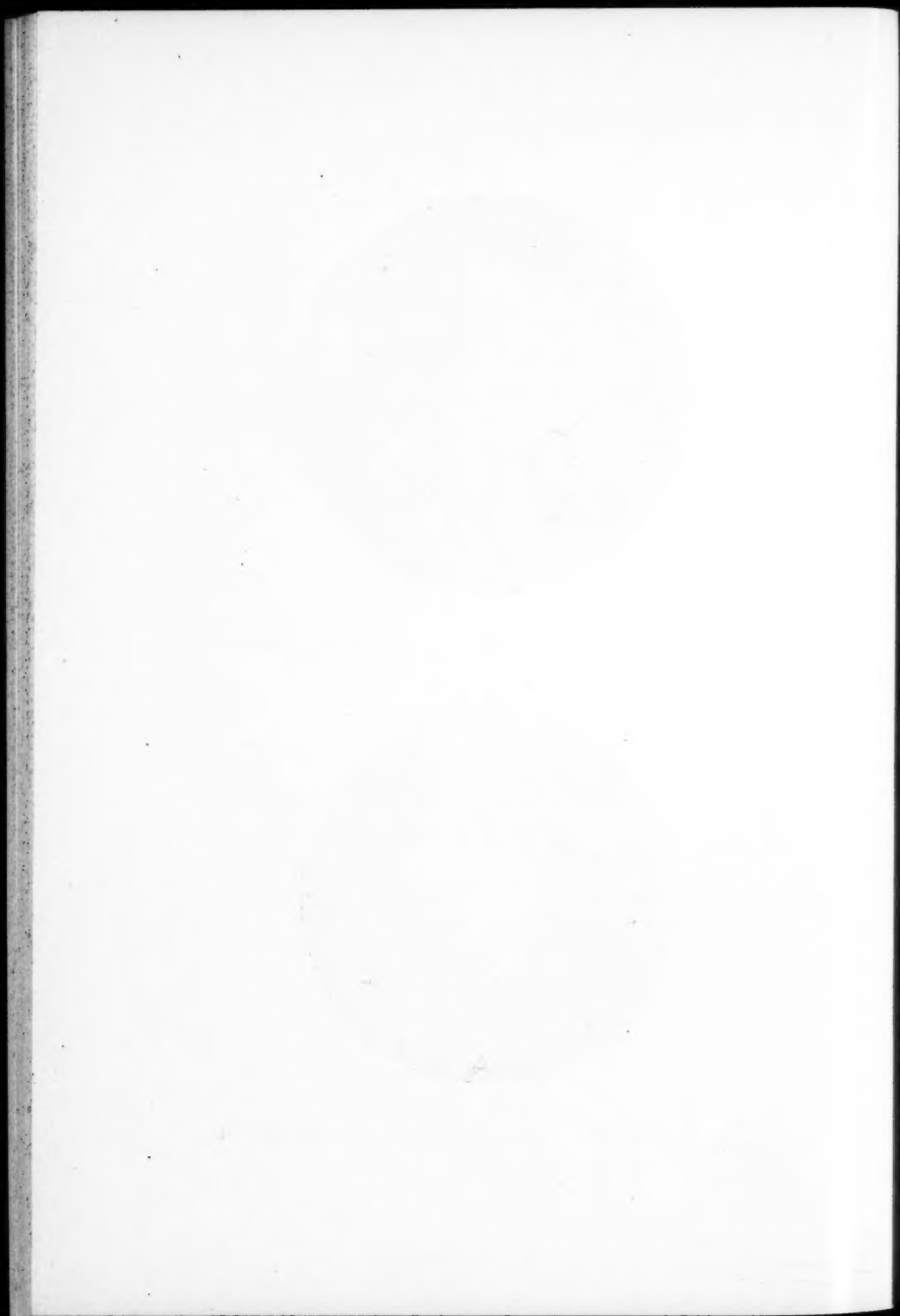


FIGURE XVII.

Same as Figure XVI, under higher power. Showing epithelial lakes.



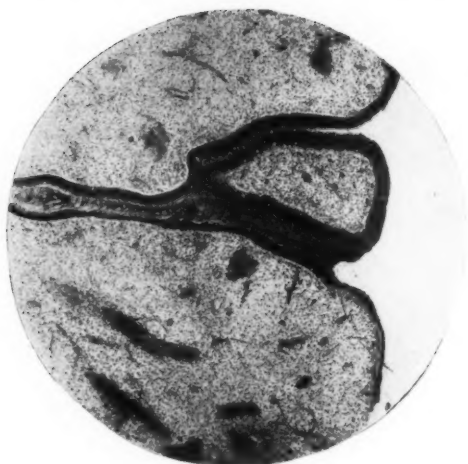


FIGURE XVIII.

Hypertrophy of the inferior turbinate body. Inflammatory edema. Marked thickening of the epithelium with folded-in masses. Showing keratosis.

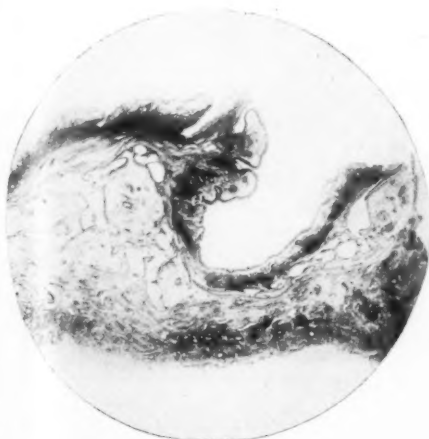


FIGURE XIX.

Papillary hypertrophy of the inferior turbinate body, showing bone and cartilage hypertrophied, with considerable rarefaction.



FIGURE XX.

Same as Figure XIX. Very marked thickening of the epithelium of the papilla.





FIGURE XXI.

Chronic intumescence of the inferior turbinate body. Marked inflammation of the surface epithelium. New blood vessels. Almost complete atrophy of all the glands.

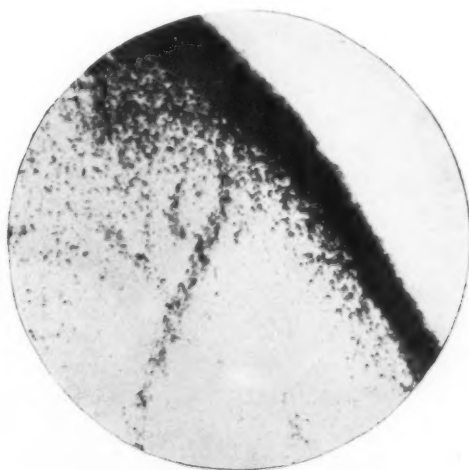


FIGURE XXII.

Same as Figure XXI, only higher power.





FIGURE XXIII.

Inferior turbinate in atrophic rhinitis (chip removed experimentally). Showing metoplasia of epithelium of the median side and thickening of the antral side. Mucous glands are still present, although distended.

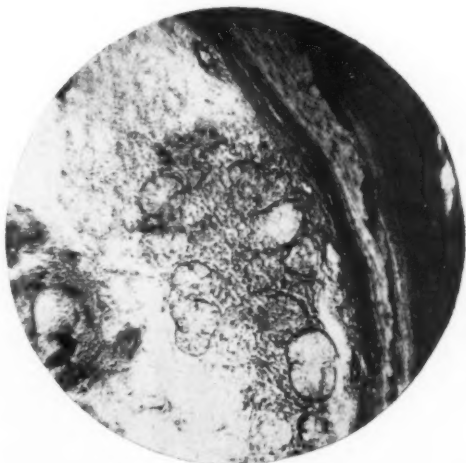
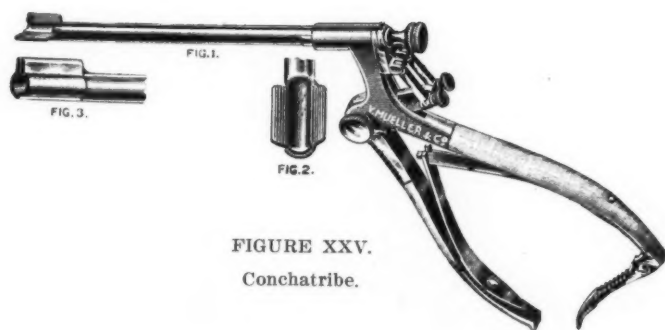


FIGURE XXIV.

Same as Figure XXIII under higher power. Shows gland distended. The bone appears normal in structure.







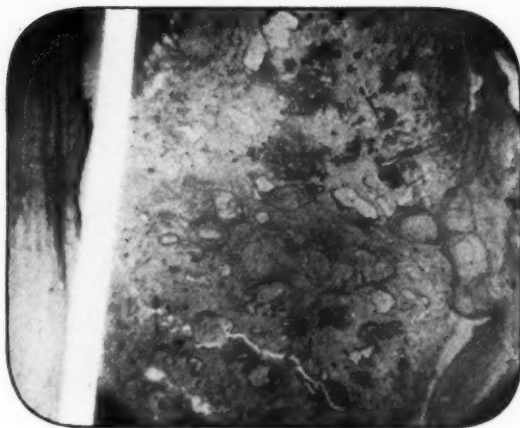


FIGURE XXVI.

Crusting of hypertrophied inferior turbinate (concha tribe). Removed on third day. Showing: (1) Marked round cell infiltration. (2) Areas of necrobiosis.



FIGURE XXVII.

Same case as Figure XXVI. Removed on tenth day. Showing tissue practically all shrunken and little evidence of organized tissue.





FIGURE XXVIII.

Luetic exudate, principally fibrin. Very few leucocytes and round cells.



FIGURE XXIX.

Apparently true myxomatous polyp of the inferior turbinate. Rarefaction of the bone; inflammatory edema of the soft parts.





FIGURE XXX.

Eschar following cauterization of the inferior turbinate. Showing marked leucocytic infiltration arranged in small masses on the margin. The pathologic change of the turbinate was chronic interstitial hypertrophy.

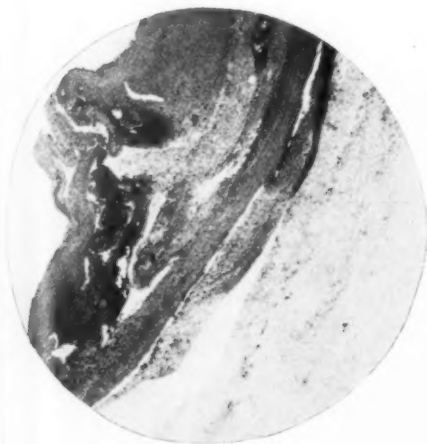


FIGURE XXXI.

Eschar of the inferior turbinate following cauterization. Showing fibrinous organization with papillary formation. Marked leucocytic infiltration. The pathologic condition of the turbinate was vascular hypertrophy.



FIGURE XXXII.

Eschar following cautery eleven days after. Showing involution of the epithelium and covered with very thick, somewhat homogeneous exudate. Some fresh blood. The pathologic change of this turbinate was epithelial hypertrophy.



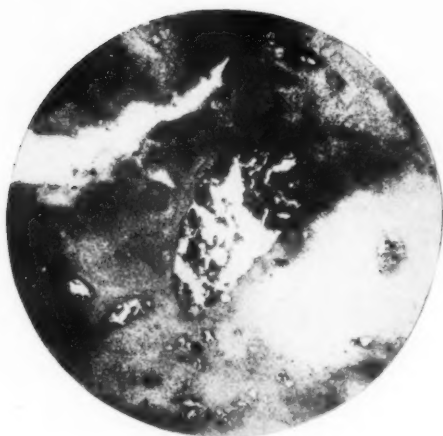


FIGURE XXXIII.

Exudate from nasal cavity following cautery of the inferior turbinate and bismuth injection as dressing. Shows marked round cell infiltration surrounding masses of bismuth.

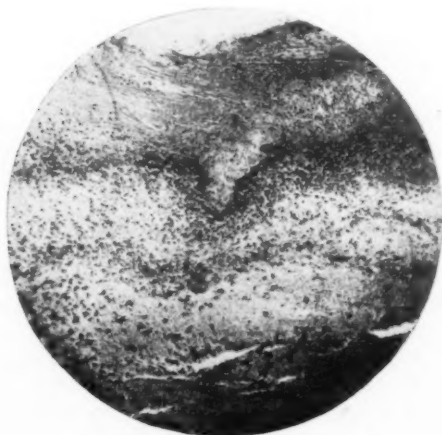


FIGURE XXXIV.

Fibrinous exudate. After ethmoid operation. Showing very marked leucocytic infiltration and fibrinous formation. Leucocytic appearance larger than either cautery or in lues.



LXI.

THE SOCIOLOGIC ASPECT OF DEAFNESS, CON-
GENITAL OR ACQUIRED IN EARLY LIFE, WITH
A SUGGESTION FOR A BETTERMENT
THROUGH INDIRECT EFFORT.

By H. B. YOUNG, A. M., M. D.,

BURLINGTON, IOWA.

If it were not for the twofold fact that otologists must eventually decide upon what constitutes total deafness, and will be looked to more and more to curtail its occurrence, both by prevention and treatment, this topic might be left wholly to the consideration of educators and economists. But the rosy view of the future for this class of unfortunates, painted by enthusiasts among the educators, doubtless encouraged thereto by enthusiasts among the otologists, bids fair to create an expectation on the part of the public, which, especially if it should not be realized, will make the legitimate work of both educators and otologists increasingly difficult.

According to John Dutton Wright,¹ there are approximately fifty thousand so-called "deafmutes" in the United States, for whom the great problem is to furnish more effective means of communication with their fellowmen, and more remunerative occupations, if they are to have the more intimate relations with society which, in his opinion, is their due. Of paramount importance is the means of communication; and that also, in his opinion, means nothing less than the use of actual speech associated with lip reading. In other words, we must teach the deaf to speak and to abandon the sign language. But this presupposes, first, that there can be found a sufficient number of teachers competent to carry on such a work, plus the munificent state to bear the expense; and, second, that the deaf will respond in a measure commensurate with the effort involved.

Assuming for the present that these teachers and funds will be forthcoming, although this remains to be demonstrated (for

it is equivalent to the support of a small army), we must yet inquire about the prospects of response from the beneficiaries.

Of the thirteen thousand pupils now in the schools for the deaf (again I quote from Wright) and those in attendance for the past ten years, approximately seventy-five per cent have had oral instruction; but of these only twenty-five per cent have gone beyond the experimental stage, i. e., made practical use of it.

In the light of the estimate that twenty-seven per cent only of "deafmutes" are totally deaf, this naturally suggests two questions, viz.:

(1) Is this twenty-five per cent the intellectual limit of its application?

(2) Does it indicate the number of those who, after more or less practice, consider it an improvement on other modes of communication?

In considering the first question we must remember that total deafness means deficient brain capacity in varying grades, from just those things which would come to it through the medium of sound, to those grosser defects the sequelæ of the meningitis which is so often the causative factor. In consequence of this the seventy-three per cent who are supposed to retain "islands of hearing," and therefore more favorable subjects, may not all be eligible. In considering the second question we must estimate the influence of an artificiality in the process, recognized alike by the producer and receiver.

Makuen,² in his contribution to the symposium on "The Deaf Child," makes this statement: "Spontaneous speech development takes place only as the individual is capable of hearing speech sounds, both subjectively and objectively; and speech acquired in any other way is a forced and artificial product." To the deaf person, therefore, who objects to being in the "limelight," and that means most of them, this forced and artificial product will make small appeal; for its first effect, through unusual tone and inflection, is to make the user conspicuous.

Something like twenty years ago, while making with my colleague, Dr. Hobby of Iowa City, a systematic examination of the pupils in the Iowa School for the Deaf, the superintendent's son, himself a pupil, was exhibited as a triumph for oral-

ism. Later, in the privacy of our room, we found ourselves in accord with this impression: "Heart-rending! Were it my child, I would rather it remained forever silent." And quite recently, in a man from the Philadelphia school, with exclusive oral instruction, I experienced the same shock. When I learned that this man could hear until the age of seven years, had but recently taken up the sign language, and now spoke with reluctance, I had further confirmation of early impressions. It may be that, in the not distant future, familiarity with this peculiar speech will soften or blunt our sensibility to it; but the intelligent deaf person can hardly escape the feeling that he or she will be, at best, just a little less a curiosity than Helen Keller. Incidentally, let me here remind you that of the multitude (a multitude that would doubtless pay willingly for the privilege) who gaze with awe, and mayhap inspiration, on Helen Keller's achievements, few stop to contemplate the patience, perseverance and resourcefulness of her teacher, which is infinitely more wonderful.

The task of bringing any class of defectives up to a reasonable equality with those of unimpaired faculties must always be colossal; not only from the standpoint of scientific achievement, but as well from the indisposition of the public to render the needed assistance. In this instance the great obstacle in the way of change is the feeling that the duty is already well done. The state furnishes as good a school for the deaf child as for the hearing, and in a material way does more for it, in that it also provides food and lodging—and clothing, if necessary. In Iowa such education is compulsory. The idea, too, that the deaf child must have an exclusive oral environment—absolutely barred from the language of signs, involving so much additional expense—may be met with skepticism; and for these reasons: (a) Every parent uses signs as a means of teaching the hearing child to speak. (b) The good preacher, good orator and good actor (and who of you has never been a devotee at the shrine of Punch and Judy?) is distinguished from the indifferent ones of his class by his ability to press his points with appropriate gestures and signs often more expressive than words. (c) Now that the Tower of Babel, with its "confusion of tongues" is again a reality, through the advent of thousands who speak, but not in our

language, it has become a necessity to use the language of signs extensively in all the avenues of industry. (d) From time immemorial the deaf have been educated by a system of signs; in most schools are so educated today; and the majority of those who have acquired speech make more use of the signs. (e) In short, when 89,950,000 people are using the sign language, more or less, every day, it is hard to imagine a condition in which the 50,000 scattered broadcast, can be shut out from it.

The sign language, therefore, however much it may interfere with the development of oralism, is here to be reckoned with. Nor is it necessarily a relic of barbarism. At the recent Congress of the Deaf in Paris, a world's congress with educated people from many lands, the sign language prevailed and was found adequate for a general interchange of ideas.

As a matter of fact, everybody knows, or should know without such evidence, that the sign language is the only universal language. It seems, however, to be no less a fact that the general conception of it is wholly inadequate. Even De l'Epee, whose name is inseparably connected with its best known usage, had a singularly narrow view of its merits. Volapuk, which had its brief day, et id omne genus, were attempts at a universal language of words—a superhuman and, in view of the possibilities of signs as conveyors of ideas regardless of words, a gratuitous task. De l'Epee and his colleagues seem not to have risen above this weakness. Provision for words gave existence to the manual alphabet.

The real sign language, however, has to do only with subject, object and action, leaving each race to give them such word expression as it will; and may profitably be taught to the hearing as well as the deaf.

It must also be acquired just as any other language is acquired; and on this account it is best to start with it in childhood, before there is a definite knowledge of the construction of spoken language. This means, before grammar is taught. Some of us know, better or worse, the French, German, Italian, Spanish, etc., but only those of us who had such environment in childhood can express ourselves in these languages without that awkwardness which imperils our intelligibility to those of the mother tongue. Thus it is probable

that none of us would ever become proficient in the sign language; but our children or grandchildren might become facile to a surprising degree, and that without a manual alphabet. For the manual alphabet need be only an incident, just as we spell out these technical expressions to the stenographer who might not transcribe them correctly.

When it is realized that one may think in signs the same as in words, which those skilled in their use really do, it may even be a matter of surprise that the sign language was ever considered a makeshift, and there were few to do it reverence.

As a basis for that systematic arrangement which is essential for the study of any language, we already have the so-called natural signs, which are practically common to all peoples. From these infinite elaboration is possible; but for the average person a thorough familiarity with these alone may be sufficient. The English language is composed of many thousand words, but the average person with fair education seems to get along with at best a vocabulary not exceeding two thousand; and a single sign may be equivalent to several words—sometimes more expressive.

That many desirable results could accrue in a general acquisition of the sign language seems almost beyond question. For present purposes, however, it is sufficient to point out:

(1) That through the element of personal profit in it, its teaching would command public interest and support.

(2) That through a common means of communication between the hearing and the deaf child they would be brought into closer relationship.

(3) That through this closer relationship there will naturally follow an appreciation of the blessing of hearing and the curse of deafness, now known only to those under the curse and those besought to lift it—an appreciation which will foster that sympathy so aptly described by Dr. John Brown of Edinburgh as the motive and not the emotion.

This argument, while in the main a plea for general instruction in the sign language for the purposes set forth, has also a medical bearing. Were it germane to the subject, I would make some observations on the difficulties attendant

upon the determination of the degree of deafness in the deaf-mute, founded upon the examination, physical and functional, of the pupils in the Iowa School for the Deaf, to which I have referred; and how I thought I discovered that, in those with negligible drum change, the remnants of sound perception were mostly to be found in the left ear. But under the title, matters of policy alone may be appropriately considered.

The brilliant work of Wright and a few others who, like him, are advocates of oralism pure and simple, gives us, as otologists, a new question to consider when we are consulted about the management of the deaf child. Some of our number have already committed themselves as endorsers of this method for the child who possesses "islands of hearing," especially those within the "Bezold scale"; and they are men of standing, whose influence may go far with those who have limited association with this class of children. I cannot but think that this endorsement, if allowed to go unchecked, will work misfortune to the otologist and deaf child alike. It is not a question whether the deaf child can be made to speak. Undoubtedly the great majority of them can—some of course much better than others. It is rather a question whether the deaf child's welfare and happiness will thereby be measurably enhanced; and this has not yet been proven. However natural our expectations that oralism would do much that is claimed for it, be worth the added cost, we find, even in the last ten years with its largely increased practice, only sporadic realization of these expectations. It is hardly thinkable that this can be explained on any ground but natural causes, mostly beyond the range of human effort; and, on this account, it becomes our plain duty to warn the parents of deaf children against too great expectations, pointing out as far as may be the obstacles peculiar to the physical and mental make up of the individual and those dependent upon social conditions. For the latter I know of nothing more comprehensive than the declaration of the lady from whose letter Dr. Gallaudet quoted in his contribution to the symposium previously referred to. Every otologist should have a copy of this on his desk. (See Appendix.)

For the betterment of this social condition the general acquisition of the sign language, which I here advocate, may

be looked upon as speculative; but from a well known teacher of the deaf, one who has spent practically his whole life among them, I have the assurance that such a course¹ would be hailed with acclaim by the deaf.

APPENDIX.

In a recent letter a deaf lady of high social standing and unusual mental ability, who was educated in an oral school, writes as follows:

"It seems to me the pure oral teachers expect too much of both the deaf and the hearing. They think that the former should be capable of an equality with the latter, which is physically impossible. They think the hearing should receive the deaf with open arms, or at least meet them half way. They ought to, of course, but the practical question is, Do they? In most cases, no. Where there are deaf friends or relatives, something of interest and kindness will be shown by the hearing, but with ordinary people the deaf are simply strange creatures, like the idiotic or insane, though of course in a less degree. The great majority of oralists are absolutely ignorant of the way they are laughed at behind their backs. I myself knew nothing of this while I had home and family to ensure me respect, but I have had some bitter experiences since then.

"For this reason, if for no other, those with bad voices should not be forced to talk. They simply make themselves a laughing-stock among the hearing. I have been told that my voice was not specially disagreeable, yet I have known hearing friends to pass me on the street without recognition, and when I demanded an explanation, confess that they did not wish the friends they happened to be with to hear me speak. Is not that enough to seal the lips of any sensitive oralist?

"In all this I am putting myself in strong antagonism to my school, but it is not to be helped. Truth and common sense should be considered as well as theory, and with the theories of the pure oralists I cannot agree.

"I insist on signs and signs only in public speaking to the deaf. On March 20, 1910, I was present at the confirmation service at Trinity Church, Boston, where Mr. Smearing inter-

preted to us the sermon of Bishop Lawrence. When I came to read the printed report of that sermon I found nothing new. Had I been seated with the general audience, I should not have known a word from beginning to end."

REFERENCES.

1. Wright, John Dutton: Address to New York Association of Physicians. Volta Review.
2. Makuen: Laryngoscope, June, 1910.

LXII.

ETIOLOGY AND PROPHYLAXIS OF TUBERCULOUS
LARYNGITIS.

By JULIUS DWORETZKY, M. D.,

OTISVILLE, NEW YORK.

I wish to submit to you a report of my work in the Nose and Throat Department of the New York City Sanatorium, at Otisville, New York. This report is based on the findings upon examination of six hundred patients, with special reference to one hundred and twenty-eight cases of laryngeal tuberculosis. Dr. W. L. Rathbun, physician in charge, requested me to write a paper on some of my work in addition to this report. Since the larynx is so readily prone to tuberculous infection, I decided upon "Laryngeal Tuberculosis" as the topic for the evening. However, as it is practically impossible to give a fair presentation of the subject in the short time at our disposal, I have chosen the "Etiology and Prophylaxis of Laryngeal Tuberculosis" as the title for my paper, leaving the pathology, diagnosis, symptomatology, and treatment for future presentation.

It is generally supposed that every specialist is inclined to overrate the specialty that he practices. I am in no position to argue this point, but I do wish to say that too great importance could never be attached to the specialty of the nose and throat, particularly when practiced in an institution of this character. Out of five hundred patients examined at the Otisville Sanatorium, laryngeal tuberculosis was diagnosed in one hundred and twenty-eight, or 25.6 per cent of the cases.

Laryngeal tuberculosis is a very sad and unfortunate complication of pulmonary tuberculosis. To emphasize this fact I wish to give you the opinion of some of our most eminent authorities. Brown claims that the average duration of life of a patient afflicted with chronic pulmonary tuberculosis is about eight years. The average duration of life of a

patient suffering from laryngopulmonary tuberculosis is a great deal shorter. Sir St. Clair Thompson believes that tuberculosis of the larynx renders the prognosis of pulmonary tuberculosis twice as gloomy; and in the majority of cases it is incurable if not treated promptly. Most authorities give it from two to three years. This difference in length of life cannot easily be overlooked, and to me it seems that an affection of this character undoubtedly deserves close attention and careful study. Only then may we hope in the future to be able to lessen the frequency of occurrence of this grave complication.

Modern scientific medicine is largely based on the science of prophylaxis. Prophylaxis has been producing most wonderful results in reduction of mortality from pulmonary tuberculosis. In New York City alone there has been a decrease of forty per cent in mortality from tuberculosis between 1887 and 1902, a period of fifteen years. (Baldwin, *Osler Modern Medicine*.) To combat a disease with prophylactic measures is the most scientific, the most reasonable, and the most economic way. Could not prophylactic measures be taken to combat laryngeal tuberculosis as well? A great deal has been said about the treatment of laryngeal tuberculosis, medical as well as surgical, but very little has been spoken of the preventive treatment of this affection. There surely must be some reason or reasons for one patient developing laryngeal tuberculosis, while another with similar pulmonary lesion and apparently similar general condition is entirely free from clinical evidence of this disease. A question of this kind is of utmost importance and undoubtedly deserves unlimited consideration.

A successful struggle against a disease on preventive lines requires one to become thoroughly familiarized with all the factors concerned in the causation, and for this reason I intend to devote some of our time to the discussion of the "Etiology of Tuberculous Laryngitis."

Tuberculous laryngitis is undoubtedly secondary to tuberculosis elsewhere in the body. Primary tuberculosis of the larynx is of very rare occurrence. In an institution like ours it is no more than reasonable to assume that tuberculosis of the larynx is entirely secondary to that of the lungs; the direct cause being the tubercle bacillus. Out of six hundred pa-

tients, one hundred of which were children, one hundred and twenty-eight adults were found to have laryngeal involvement. No tuberculosis of the larynx was detected in any of the children. From this it would seem that tuberculous laryngitis is, for some reason or other, an affection mainly of adult life.

I also found that 28.8 per cent of the cases of tuberculous laryngitis were in the male, while only 20.7 per cent were in the female. Thus it would appear, from this study, that it is more common among male patients. On further study I noticed that one hundred and sixteen, or 91 per cent, of the patients were cases with positive sputum, a fact which is worth while noticing. Of the twelve negative cases, seven were of doubtful diagnosis as to the laryngeal lesion, three have not had a sufficient number of sputum examinations, while two had definite lesions with a persistent absence of bacilli in the sputum. Bullock, in his paper read before the Sixth International Congress on Tuberculosis, reports one hundred cases of tuberculous laryngitis, 25 per cent of which were ulcerative, and 75 per cent nonulcerative. He found a positive sputum in every case, a fact which almost agrees with our findings here.

I should next like to consider the mode of invasion of the larynx by the tubercle bacillus. Authorities still disagree. Heinze, Coakley and others believe the lymphatics to be the most common route of infection. Bonney, Ballenger and others regard the sputum from the lungs as chief conveyor of the infection. My observations here bear out the latter theory very well, by reason of the fact that only 9 per cent of our cases of tuberculous laryngitis have been negative, and most of these were either of doubtful diagnosis, or else did not have a sufficient number of sputum examinations. Of course, it might be claimed that the bacilli come from the ulcerations of the larynx; but then how would we account for the positive sputa in 62 per cent of the tuberculous laryngitis cases where no ulcerations were present? Therefore, it would really seem that a positive sputum is an "essential," or at least "one" of the essential factors in the development of tuberculous laryngitis.

Seventy and six-tenths per cent of adults of this sanatorium have or have had a positive sputum, while only 25.6 per cent of the adult patients have laryngeal tuberculosis.

The question would naturally arise, "Why do only 25.6 per cent of positive patients show clinical laryngeal tuberculosis, while the remaining 45 per cent of positives are apparently free? To me it seems that a correct answer may solve this gigantic problem and tell us how and why tuberculosis of the larynx is contracted. After having learned this we may be able to apply prophylactic measures, and thus indefinitely prolong hundreds of human lives.

I carefully talked this matter over with Dr. Rathbun, and upon his recommendation I proceeded to examine the nose, throat and larynx of every patient in this sanatorium, carefully noticing and classifying the pathologic conditions. Of the six hundred patients examined, I found that only two hundred and twenty-six larynges presented a normal appearance. The remaining three hundred and seventy-four were abnormal. Out of the latter, two hundred and forty-six showed signs of congestion and hyperemia without any local infiltration, and one hundred and twenty-eight showed more or less tuberculous involvement. This high percentage of catarrhal condition of the larynx suggested itself to me as a probable predisposing cause. As I just said, almost in every case we have an open pulmonary lesion which means a constant discharge of bacilli flowing over and bathing the laryngeal mucosa. The anatomy of the interior structures of the larynx is very complex, and pus traveling over it is bound to become stagnated and in that way adhere to the minute folds and creases. Healthy mucous membrane may be able to resist it by its natural immunity, but when inflamed, besides the minute abrasions that may be present as a result of it, the intercellular spaces become widened, and the bacilli, not meeting with any resistance, enter the mucosa and submucosa, setting up a tuberculous inflammation.

With the above theory in view as a probable *modus operandi*, I carefully questioned, examined and reexamined one hundred and twenty-eight cases of tuberculous laryngitis, and then classified them as to age, sex, sputum, pulmonary lesion, nasal obstruction or disease, inflammation of pharynx, tonsils and soft palate, history of "colds," excessive use of voice, cough, use of tobacco and alcohol. The findings were most interesting, and on account of their important bearing on the subject I wish to give them to you in detail.

Age.—All cases of tuberculous laryngitis were found in adults, most of them between the ages of twenty and forty-five. No tuberculous laryngitis was detected among one hundred patients below fourteen years of age. This may partially be explained by the fact that only 5 per cent of the children have had a positive sputum. It would be hard to account for the absence of tuberculous laryngitis in children, if we were to accept the lymphatics as the most common route of invasion, for children are very prone to tuberculosis of the bronchial and cervical glands.

Sex.—28.8 per cent of tuberculous laryngitis occurred in males, while only 20.7 per cent occurred in females. This may be accounted for by the fact that 80 per cent of the males had a positive sputum, while in the females it was present only in 56 per cent. Also by the fact that men use tobacco and alcohol, while their use among women is so limited that we may disregard them as factors. Men are also more subject to exposure.

Sputum.—One hundred and sixteen, or 91 per cent, of the adult patients with tuberculous laryngitis have or have had a positive sputum. Out of the remaining twelve patients, seven are still of doubtful diagnosis as to the laryngeal lesion, three have not had a sufficient number of sputum examinations, while two were persistently negative.

Nasal obstruction or diseased condition of nasal chambers, in my mind undoubtedly acts as predisposing factor. As you all know, the nose, outside of the special function of smell, has two other very important functions. First, is the function of respiration. The air as it goes through the nose, before it reaches the larynx and the lungs, becomes modified. The outside air is very seldom of the proper temperature to enter the lungs, and for that reason as it passes through the nasal chambers it is warmed by the blood circulating through the mucous membrane of the nose, and in that way made fit for assimilation in the lungs. Besides being warmed the air also is moistened. This function belongs to the mucous secreting glands and the "swell bodies" of the inferior and middle turbinates. It has been estimated that about one pint of serum is thus transferred from the nasal mucous membrane to the lungs in twenty-four hours. The nose also acts as a filtering medium by means of the hairs in the vestibule and the nasal mucous lining. The latter with its moist surface

catches the very fine particles of dust which may have escaped the hairs. The warming and moistening of the air is absolutely essential for the protection of the delicate structures in the lower part of the respiratory tract from the great variation in extreme temperatures.

Another and very important function, as claimed by Ballenger, is that of ventilation of the nasal accessory sinuses. As you are aware, all the accessory sinuses are in direct communication with the nasal chambers through ducts and channels, and any interference with the ventilation of the sinuses, as by a growth, enlarged middle turbinate, etc., will cause a lowering of resistance of the lining mucous membrane, while the diminished amount of oxygen will allow the secretion to undergo rapid decomposition.

With these two above described functions in mind, we can easily see how a deviation from the normal anatomy will cause interference with above functions, i. e., respiration and ventilation of the accessory sinuses, and subsequently produce a pathologic condition.

After having briefly considered the function of the nose, I wish to take up the pathogenesis of nasal obstruction. Ninety-two per cent of the cases of tuberculous laryngitis were found to have nasal obstruction or disease; 36.7 per cent having slight obstruction; 18 per cent having moderate obstruction; 8 per cent marked; and 29.3 per cent suffering from either an atrophic or suppurative condition of the nose. Ballenger claims a deflected septum as one of the great predisposing factors to all kinds of rhinitis. At this sanatorium, 60.8 per cent of all the patients present more or less abnormality of the septum.

When a septum is deflected, one chamber is naturally larger than the other and more air will therefore circulate through it. With an increase in the amount of inflowing air, there is an increase in function of the turbinates of that side, and therefore a compensating hypertrophy. At first the hypertrophy is of the soft or turgescient type, which consists merely of an engorgement of the venous plexuses of the "swell bodies" of the turbinates. But in the course of years, if this condition is not remedied, a real hypertrophy of the turbinates may occur, on account of the hyperemia and consequent increased nutrition. If the obstruction exists higher up in the neighborhood of the middle turbinate, the retention and de-

composition of the secretion in the superior meatus and the posterior ethmoidal cells will cause a prolonged low grade irritation which will result in hyperplasia of the turbinates, causing a hyperplastic rhinitis, which is characterized by an increased formation of connective tissue cells. We are all familiar with the tendency of connective tissue to contract. As time goes on, the connective tissue will keep on contracting, thus cutting off the blood supply and choking the glandular structures. The subsequent result is atrophy with formation of scabs. Of course, the above, although true in many instances, does not necessarily mean that every case of deflected septum will cause a turgescent rhinitis which will finally terminate in the atrophic form. The pathologic process may be arrested at any point. Some cases can be averted by medical or surgical means, a great many will be checked by nature, while the rest will probably take the course as outlined above.

An abnormal condition in the nose will eventually produce a mild inflammatory condition in the larynx, due to the irritation caused by the improperly modified air. Patients with nasal obstruction finally become partial or total mouth breathers, and in that way deprive the inspired air of the proper amount of warmth and moisture. Those suffering from an atrophic condition, in spite of the patency of the nasal chambers, likewise do not receive properly modified air, on account of the destruction and atrophy of the mucous membrane with its venous plexuses and glands.

So much for abnormal nasal condition as a predisposing cause to tuberculosis of the larynx.

Pharyngitis may be considered as another predisposing factor to tuberculous laryngitis. Seventy-nine per cent of the cases of tuberculous laryngitis were found to be suffering from chronic pharyngitis. Chronic pharyngitis is mainly due to improper breathing, and is therefore considered as secondary to chronic rhinitis. The mucous membrane of the pharynx is continuous with that of the larynx, and there is no reason why a chronic inflammation of one should not extend to the other. Then again, we are all familiar with the fact that all patients with pharyngitis or nasopharyngitis have a persistent desire to clear their throats. They usually do it by hawking and coughing, an act which by itself would tend to cause a congestion of the larynx. The mucoid or mucopurulent secretion acts as a mild irritant, producing chronic congestion

and inflammation and a constant desire to cough and expectorate. Personally I believe that a great proportion of the sputum of the patients of the sanatorium is nothing more than the secretion from the nasopharynx, and by treating the latter the amount of sputum could be greatly reduced in many instances.

Tonsillitis and Diseased Condition of the Soft Palate.—Sixty per cent show a diseased condition of the tonsils or soft palate, which means nothing more than repeated attacks of acute inflammation. It is no more than reasonable to assume that in many instances of tonsillitis the inflammation also extends to the laryngeal mucosa, thus with each attack lowering its resistance.

Subject to "Colds."—Sixty per cent of the cases give a history of being very susceptible to "colds." This naturally suggests many previous attacks of acute rhinitis, pharyngitis, and tonsillitis. One attack followed by others usually leads to chronicity, which merely confirms the above statistics.

Overuse of Voice.—Thirteen per cent of the cases give a history of excessive use of voice—like teaching, singing, proof-reading, etc. We can all easily see how this will cause congestion, chronic hyperemia and lowered local resistance of the larynx.

Cough is another very important factor. One hundred and twenty-six, or 98.5 per cent, of the cases give a history of cough extending over a period of from a few months to many years. Although most of the coughing is probably of pulmonary origin, we must not forget that a cough may also be due to some abnormality in the nose, pharynx, or larynx. Continuous coughing will in time cause a congestion of the larynx and later produce a catarrhal condition. At this stage it is evident enough how the cough can become laryngeal and in that manner complete the vicious circle. Personally I believe that a great deal of the coughing is due to some irritation in the nose, pharynx, or larynx, and by local treatment the cough could be greatly diminished.

I can distinctly recall the case of a patient with a third stage pulmonary lesion complaining of excessive cough, especially at night on lying down, so that he could hardly gain any sleep. Upon examination I found a markedly elongated uvula resting on the dorsum of his tongue, even with the tongue depressed. I amputated the uvula, and the very same

night the patient rested most comfortably without even coughing once. His sputum also became decreased to half of the previous amount.

Tobacco.—Smoking may also be looked upon as a contributing factor. We are all familiar with the effects of smoking on the pharynx. Is there any reason why a similar condition should not occur in the larynx? Seventy-eight and five-tenths per cent of the male patients give a history of continued use of tobacco, 40 per cent having used it to excess. According to our statistics here, tuberculosis of the larynx is about one and one-half times as common in the male as in the female, and smoking would naturally suggest itself as one of the reasons for this discrepancy.

Alcohol may play a prominent rôle as a predisposing etiological factor. It may act directly by causing irritation of the neighboring organs, or indirectly through the circulation. Forty-two per cent of the men give a history of the use of alcohol, many of them to excess. This, too, may be one of the reasons for the greater frequency of tuberculosis of the larynx among the males.

Last, but not least, I wish to consider the general condition of the patient as a predisposing factor. Most authorities agree that about 90 per cent of the people have more or less tuberculosis in some part of their system, and it is only on account of lowered vitality and superinfection that some of these develop clinical tuberculosis. The same is undoubtedly true of tuberculosis of the larynx. Incipient cases are more resistant to laryngeal tuberculosis than the more advanced cases. The reason for this is that in incipient cases the general resistance is usually good as compared with that of the more advanced cases, and the infection itself is not as overwhelming as in cases of more or less advanced tuberculosis. Upon examination of five hundred adult patients, I found that tuberculous laryngitis was present in 12 per cent of the incipient cases, 26.4 per cent of the moderately advanced, and 45.3 per cent of the advanced cases.

In summing up the above, I wish to say:

First, that tuberculosis of the larynx is apparently caused by the direct invasion of the mucous membrane of the larynx by the tubercle bacillus. If the lymphatics were the main channel of infection, then why is tuberculosis of the larynx so rare or almost absent in closed pulmonary lesions?

Second, that a predisposing cause is almost as essential as the direct cause; and anything that tends to congestion and chronic catarrhal inflammation of the larynx should be considered as a predisposing factor. If only the bacillus tuberculosis were necessary for the causation of clinical tuberculosis of the larynx, then why do not all patients with positive sputa develop tuberculous laryngitis?

PROPHYLAXIS.

Now I wish to briefly take up the prophylaxis of tuberculous laryngitis. As I mentioned early in my paper, much has been written on the curative treatment of laryngeal tuberculosis, while the prophylactic phase has apparently been neglected. In speaking of the actual treatment, all authors, without exception, agree on the absolute necessity of vocal rest in addition to the hygienic and the dietetic treatment. Besides the above, most of them also believe in the medical or surgical treatment. Bullock is a great believer in the efficiency of formalin solution in the treatment of tuberculous laryngitis. Minor advocates the use of iodoform. Other authorities advise the use of lactic acid and other astringents. The electric cautery has been used by many with very good results. Pettit claims that he healed from 70 to 85 per cent of incipient laryngeal tuberculosis by the cauterization of the diseased tissues. Wood claims that his experience with the electric cautery in laryngeal tuberculosis has led him to almost discontinue any other method of treatment. Arnoldson, in the *Journal of Progressive Medicine*, believes that medical treatment has little influence on the course of the disease, and advocates endolaryngeal surgical measures. Tuberculin has been used with varied success.

So far no specific remedy has been discovered for this affection. Personally I believe that different modes of treatment are applicable to the different phases of the disease. Some early cases will undoubtedly do well under general treatment and vocal rest. In cases of soft granulating ulceration, formaldehyd seems to be of some value.

Cases that have advanced considerably will very seldom yield to any local remedy, and the expectant treatment and the treatment of the symptoms as they arise are about the only things that can be done in their behalf.

However, we have learned that tuberculous laryngitis is a secondary disease, occurring mainly in adults with positive sputum; furthermore, we may assume that tuberculosis of the larynx, like tuberculosis elsewhere in the body, has a direct causative factor, i. e., the tubercle bacillus and predisposing factors like those enumerated above. Therefore, we face the following question: "Are we justified in adhering to the hitherto generally adopted policy of merely treating the disease as it arises, without making an earnest effort at prevention?"

To prevent tuberculosis of the larynx we must, first, combat and check the direct cause. Second, remove the predisposing factors.

To accomplish the first, we must treat the patient's pulmonary lesion and also improve his general condition. Our methods of treatment at the Otisville Sanatorium fully cover this point. Our constant aim is, on the one hand, to check the activity of the pulmonary lesion by endeavoring to promote fibrosis; and, on the other hand, to raise the patient's general resistance and render him more or less immune.

This general treatment, although most excellently applied and carried out at our sanatorium, does not very often accomplish the most desired purpose, i. e., the permanent healing of open lesions. Therefore, not being able to remove the direct cause, the only and most logical thing to do would be to raise the local resistance of the larynx.

Here I think it in place to briefly mention the routine adopted by the throat department of our sanatorium. Each patient on admission has to undergo a thorough nose, throat and ear examination. The findings are carefully noted.

Recently we have instituted a system of books in which the patients are classified as to the previous history, age, sex, habits, pathologic findings, symptoms, etc. After carefully examining the patient we are in a position to advise him of any detected abnormality which later may have some influence on his larynx. A deflected septum causing more or less trouble is of common occurrence. Most recent authorities seem to regard a deflected septum as the underlying factor of many pathologic conditions. A deflected septum with consequent obstruction in respiration or drainage should be corrected. The modern submucous resection is a most excellent operation, and in the hands of a skillful rhinologist most always

meets with success. The operation can be easily done under local anesthesia, and by taking the proper precautions there is not much loss of blood. The average patient can stand the operation easily without subsequent ill effects.

Many patients will be greatly benefited by the removal of an hypertrophied middle turbinate, which very often obstructs the drainage from the accessory sinuses. The operation is very simple also and can be done under local anesthesia.

Cases of obstructed nasal breathing due to enlarged inferior turbinates should be carefully studied before resorting to any particular method of treatment. We must always remember that our aim is not only to obtain patent nasal chambers, but also to preserve the condition of the mucous membrane of the nose as nearly normal as possible. The inferior turbinates contain most of the "swell bodies" of the nose, and by removing them we deprive the nose of its physiologic efficiency. The patient afterwards complains of dryness and irritation in the nose and neighboring organs. Most cases with enlarged turbinates have deflected septa, and by correcting the latter the turbinates later on will usually become of normal size. When the enlargement is of the turgescient type, and if a septal operation is unnecessary, the galvanocautery will give great relief.

A coexisting pharyngitis or nasopharyngitis should also be treated. Here medical treatment and the use of the galvanocautery are of great service.

Elongated uvulas with a history of excessive cough should be amputated.

Patients with diseased or hypertrophied tonsils and adenoids should be carefully questioned as to previous attacks of tonsillitis, and if such history be obtained the tonsils and adenoids should be removed.

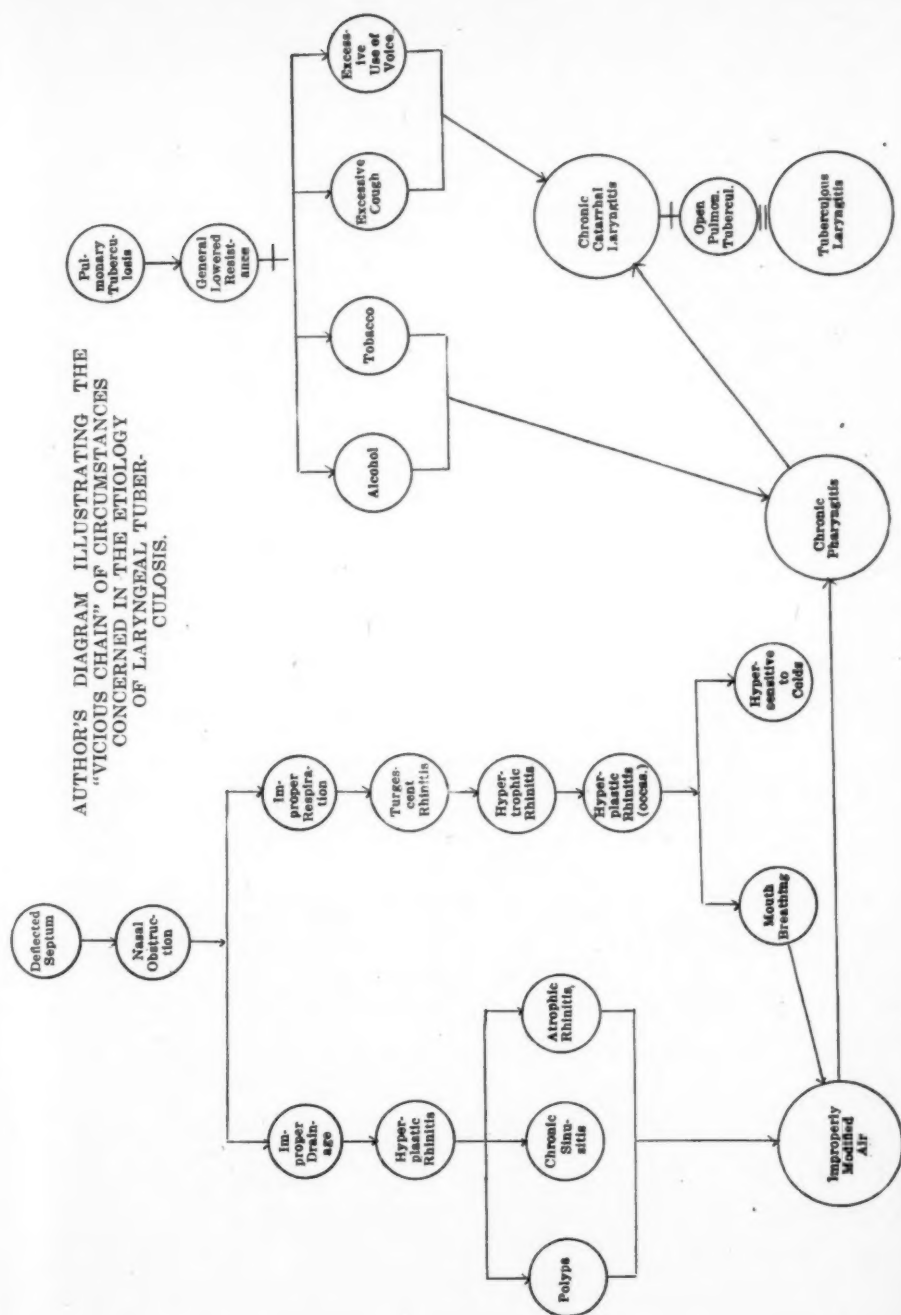
Patients with pulmonary tuberculosis, especially those with open lesions, should be dissuaded from excessive use of voice.

The use of tobacco, like the use of alcohol, should be greatly restricted. Here of course a great deal of individualization will be necessary.

Cough of pulmonary origin and not excessive is always best left alone. Excessive cough should be treated by sedatives. Cough of extrapulmonary origin should be combated by treating the underlying cause.

Finally, patients that show congestion or anemia of the

AUTHOR'S DIAGRAM ILLUSTRATING THE
 "VICIOUS CHAIN" OF CIRCUMSTANCES
 CONCERNED IN THE ETIOLOGY
 OF LARYNGEAL TUBER-
 CULOSIS.





larynx should be carefully watched and examined at frequent intervals. The congestion, although a result of morbid condition of the neighboring organs, should be treated. I find that in addition to the treatment of the neighboring organs, a spray of 5 per cent solution of menthol in olive oil has a very beneficial influence. The menthol causes slight anesthesia and a constriction of the blood vessels which is of more permanent character than that of cocain or adrenalin. It also has a soothing influence on the mucous membrane. Catarrhal laryngitis accompanied by a tracheitis is specially benefited by repeated intratracheal injections of the same solution.

By carrying out the above briefly outlined prophylactic treatment, I earnestly believe that although tuberculosis of the larynx will not be entirely eradicated, it will and it must become a complication of less frequency than at present.

I therefore enter a plea for the adoption of strenuous measures in the carrying out of the prophylactic treatment of tuberculosis of the larynx.

I have drawn up a diagram illustrating what I designate as the "vicious chain" of circumstances concerned in the etiology of laryngeal tuberculosis. On a separate sheet you will find my classification of laryngeal tuberculosis. Finally, I wish to bring to your notice a statistic report. All of these I will now pass around.

I am greatly indebted to Dr. Rathbun for his advice and many suggestions; also to the resident staff for their kind cooperation.

BIBLIOGRAPHY.

- Ballenger: Disease of Nose and Throat, Third Edition.
 Bonney: Pulmonary Tuberculosis and Its Complications.
 Bullock: Sixth International Congress on Tuberculosis, Vol. I, Part 2.
 Wood: Progressive Medicine, Vol. XVI, No. 1.
 Minor: Early Changes in the Larynx in Pulmonary Tuberculosis.
 The National Association for the Study and Prevention of Tuberculosis, 1909-10.
 Coakley: Diseases of the Nose and Throat, Third Edition.
 Pettit: Progressive Medicine, Vol. XVI, No. 1.
 Arnoldson: Progressive Medicine, Vol. XVI, No. 1.
 Brown: Osler Modern Medicine, Prognosis of Tuberculosis, Ch. VI.
 Baldwin: Osler-MacRae Modern Medicine. History and Etiology of Tuberculosis, Chap. IV.
 Lyon: Reports of Two Hundred and Forty-one Cases of Laryngeal Tuberculosis Treated at the Rutland State Sanatorium. Boston Medical and Surgical Journal, Vol. CLXXI, No. 1, July 2, 1914.
 St. Clair Thomson: Progressive Medicine, Vol. XVI, No. 3.

DEFINITION OF TERMS.

NARES.

1. *Rhinitis.*

- (a) *Turgescent Rhinitis.*—Soft or turgescent swelling of the inferior or middle turbinates.
- (b) *Hypertrophic Rhinitis.*—Causing partial stenosis of nasal chambers, hypertrophy of mucous membrane of inferior or middle turbinates.
- (c) *Hyperplastic Rhinitis.*—Increase of thickness of mucous membrane as a result of prolonged mild irritation by secretion from accessory sinuses.
Polypi.—A later result of above condition.
- (d) *Atrophic Rhinitis.*—Characterized by a sclerotic change in the nasal mucous membrane and occasionally of the underlying bone, and by presence of crusts and frequently offensive nasal breath.
Atrophic Rhinitis With Collapse of Erectile Tissue.—Not true inflammatory. Due to general anemia and characterized by atrophy of erectile tissue of nose.

SEPTUM.

1. *Deflection.*

- First Degree.*—Slight deviation from normal, without marked obstruction in respiration or drainage.
- Second Degree.*—Moderate deviation from normal, with moderate or occasional obstruction in respiration or drainage.
- Third Degree.*—Marked deviation from normal, with more or less constant obstruction in respiration or drainage.

2. *Thickening.*

- First Degree.*—Slight amount of hypertrophy of mucous membrane.
- Second Degree.*—Moderate amount of hypertrophy of mucous membrane.
- Third Degree.*—Marked hypertrophy of mucous membrane or septum.

3. *Spurs.*

- Prominent outgrowth of cartilage or bone from septum.

4. *Accessory Sinuses.*

- Sinusitis.*—Chronic inflammation of one or more of the accessory sinuses.

PHARYNX.

1. *Acute Catarrhal.*

Acute inflammation of mucous membrane of pharynx.

2. *Chronic Granular.*

Hyperplasia of mucous membrane of pharynx, accompanied by increase of size of lymph follicles.

3. *Hypertrophic.*

Thickening and hypertrophy of mucous membrane of pharynx.

4. *Atrophic Pharyngitis or Pharyngitis Sicca.*

Atrophy of mucous membrane of pharynx.

UVULA.

1. *Hypertrophied.*

Increase in size.

2. *Elongated.*

Increase in length, resting or nearly resting on base of tongue.

TONSILS.

1. *Hypertrophied.*

First Degree.—Slightly enlarged in size.

Second Degree.—Moderately enlarged in size.

Third Degree.—Markedly enlarged in size.

SOFT PALATE.

Hypertrophied Fauces.

Usually accompanies hypertrophied tonsils or granular pharyngitis.

LARYNX.

Congested.

Under which come "acute and chronic catarrhal."

• *Tuberculosis.*

Part or entire larynx presents appearance of tuberculous inflammation.

Stage A.—Doubtful stage.

Stage 1.—Stage of infiltration.

Stage 2.—Stage of ulceration.

Stage 3.—Advanced stage.

For detailed definition of stages, see "Classification of Tuberculous Laryngitis."

AUTHOR'S CLASSIFICATION OF TUBERCULOUS LARYNGITIS.

Class A.—Doubtful stage.

1. Sputum positive or negative.
2. Hoarseness in morning or persistent; may be absent.
3. Dysphagia present or absent.
4. Congestion or anemia of part or entire larynx.
5. Slight hyperplasia of interarytenoid space; may be absent. Patients of this class are repeatedly examined at intervals of two weeks. They are either discharged as cases of nontuberculous laryngitis, or, on further development of laryngeal physical signs and symptoms, are admitted to Class 1.

Class 1.—Stage of infiltration.

1. Positive sputum. Negative in exceptional cases.
2. Recurrent or persistent hoarseness.
3. Dysphagia present or absent.
4. Signs of infiltration of any or all the parts of the larynx, especially interarytenoid space, vocal cords, aryepiglottidean folds, ventricular bands, arytenoid cartilages, or epiglottis.

Class 2.—Stage of edema with superficial erosion.

1. Positive sputum.
2. Persistent hoarseness.
3. Dysphagia present or absent.
4. Dysphonia present or absent.
5. Signs of tumefaction of part of larynx with superficial erosion.

Class 3.—Advanced stage.

1. Positive sputum.
2. Marked hoarseness or aphonia.
3. Dysphagia present or absent.
4. Dysphonia present or absent.
5. Marked tumefaction and ulceration of any or all parts of the larynx.
6. Perichondritis sometimes present.*

*Hoarseness, dysphonia and aphonia are usually absent in tuberculous involvement limited to the epiglottis.

NUMBER OF PATIENTS EXAMINED.	Total.	%.
Male	332	55.3
Female	268	44.7
<i>Nares.</i>	600	600
Normal	209	34.9
Rhinitis	391	65.1
Turgescient	101	16.8
Hypertrophic	201	33.5
Hyperplastic	26	4.3
Atrophic	63	10.5
<i>Septum.</i>		
Normal	235	39.2
Abnormal	365	60.8
Deflected	188	31.3
Deflected 1 degree.....	120	20.
Deflected 2 degrees.....	50	8.3
Deflected 3 degrees.....	18	3.
Thickened	179	29.8
Thickened 1 degree.....	120	20.
Thickened 2 degrees.....	55	9.1
Thickened 3 degrees.....	4	.7
Spurs and ridges.....	47	7.8
Cartilage removed	6	1.
Ulceration and atrophy.....	11	1.8
Perforation	5	.8
<i>Pharynx.</i>		
Normal	357	59.5
Chronic pharyngitis	243	40.5
<i>Palate and Uvula.</i>		
Normal	416	69.4
Abnormal	184	30.6
Hypertrophied fauces	72	12.
Inflamed fauces	60	10.
Elongated uvula	73	12.
Inflamed and thickened uvula.....	30	5.
Cleft palate	1	
<i>Tonsils.</i>		
Normal patients	408	68.
Hypertrophied	192	32.
Hypertrophied 1 degree.....	133	22.1
Hypertrophied 2 degrees.....	46	7.7
Hypertrophied 3 degrees.....	13	2.2

THROAT TREATMENTS, MEDICAL AND SURGICAL, INCLUDING
ELECTRIC CAUTERIZATION, FOR PAST SIX MONTHS,
FROM FEBRUARY 1 TO AUGUST 1, 1914.

FEMALE UNIT.

February	59	
March	60	
April	55	
May	72	
June	120	
July	123	
Total		489

MALE UNIT.

February	80	
March	91	
April	47	
May	58	
June	90	
July	94	
Total		460
Total male and female units		949

LARYNX.

MALE UNIT.

	Total.	%.
No. of patients examined, including 25 children under 14	332	100.
Normal	105	31.7
Abnormal	227	68.3
Congested	139	42.
Tuberculous laryngitis	88	26.
Stage A	11	12.
Stage 1	53	61.
Stage 2	19	21.
Stage 3	5	6.
Total number of adult patients	307	
Percentage of tuberculous patients in male adults		28.8

FEMALE UNIT.

No. of patients examined, including 75 children under 14.....	Total.	%.
Normal	121	45.1
Abnormal	147	54.9
Congested	107	39.9
Tuberculous laryngitis	40	15.
Stage A	3	7.5
Stage 1	25	62.5
Stage 2	11	27.5
Stage 3	1	2.5
Total number of adult patients.....	193	
Percentage of tuberculous laryngitis in female adults		20.7
Average percentage of tuberculous laryngitis of all patients, male and female adults		25.6
Average percentage of tuberculous laryngitis of all patients, including 100 children under 14 years.....		21.3

INCLUDING CHILDREN UNDER FOURTEEN YEARS.

Female Patients.

Positive sputum	105	40.0%
Negative sputum	156	60.0%
	<u>261</u>	

Male Patients.

Positive sputum	227	73.5%
Negative sputum	82	26.5%
	<u>309</u>	

Total Female and Male Patients.

Positive sputum	332	58.2%
Negative sputum	238	41.8%
	<u>570</u>	

Total number of females..... 261

Total number of males..... 309

Total number of patients..... 570

Percentage of positive cases..... 58.2

Percentage of negative cases..... 41.8

NOT INCLUDING CHILDREN UNDER FOURTEEN YEARS.

Female Patients.

Positive sputum	105	56.4%
Negative sputum	81	43.6%
	<u>186</u>	

Male Patients.

Positive sputum	227	80.0%
Negative sputum	57	20.0%
	<u>284</u>	

Total Female and Male Patients.

Positive sputum	332	70.6%
Negative sputum	138	29.4%
	<u>470</u>	

Total number of females 186

Total number of males 284

Total number of patients..... 470

Percentage of positive sputum..... 70.6

Percentage of negative sputum..... 29.4

CLASSIFICATION OF 500 ADULT PATIENTS ACCORDING TO THEIR PULMONARY STAGES.*

	Male.	Female.	Total.
Stage 1.....	74	50	124
Stage 2.....	185	118	303
Stage 3.....	48	25	73
	<u>307</u>	<u>193</u>	<u>500</u>

CLASSIFICATION OF 128 CASES WITH LARYNGEAL TUBERCULOSIS ACCORDING TO THEIR PULMONARY STAGES.

	Male.	Female.	Total.
Stage 1.....	11	4	15
Stage 2.....	59	21	80
Stage 3.....	18	15	33
	<u>88</u>	<u>40</u>	<u>128</u>

*Pulmonary stages classified according to the National Association for the Study and Prevention of Tuberculosis.

FREQUENCY OF OCCURRENCE OF LARYNGEAL TUBERCULOSIS IN
THE DIFFERENT PULMONARY STAGES.

Stage 1.....	15 cases out of 124	12.0%
Stage 2.....	80 cases out of 303	26.4%
Stage 3.....	33 cases out of 73	45.3%

STATISTICAL CLASSIFICATION OF 128 CASES OF LARYNGEAL
TUBERCULOSIS AS TO ETIOLOGIC FACTORS.

	Male Unit.		Female Unit.		Total.	
<i>Patients.</i>	No.	%.	No.	%.	No.	%.
Total number	88	69.	40	31.	128	100.
<i>Sputum.</i>						
Positive	81	92.	35	87.5	116	91.
Negative	7	8.	5	12.5	12	9.
<i>Age.</i>						
Below 14.....	0	0	0	0	0	0
Above 14.....	88	100.	40	100.	128	100.
<i>Pulmonary Stage.</i>						
Stage 1.....	11	12.5	4	10.	15	11.5
Stage 2.....	59	67.	21	52.5	80	62.5
Stage 3.....	18	20.5	15	37.5	33	26.
<i>Laryngeal Stage.</i>						
Stage A.....	13	14.5	3	7.5	16	12.5
Stage 1.....	54	61.	25	62.5	79	61.5
Stage 2.....	13	14.5	11	27.5	24	19.
Stage 3.....	8	10.	1	2.5	9	7.
<i>Nasal Obstruction.</i>						
Total number.....	82	93.	36	90.	118	92.
Slight	33	37.5	14	35.	47	36.7
Moderate	13	15.	10	25.	23	18.
Marked	9	10.	1	2.5	10	8.
Atrophic and sup'tve.....	27	30.5	11	27.5	38	29.3
<i>Pharyngitis.</i>						
Total number.....	75	85.	26	65.	101	79.
<i>Hypertrophy and Inflammation of Tonsils and Soft Palate.</i>						
Total number.....	59	67.	18	45.	77	60.

	Male Unit.		Female Unit.		Total.	
<i>Subject to "Colds."</i>	No.	%.	No.	%.	No.	%.
Total number.....	50	56.	27	67.5	77	60.
<i>Overuse of Voice.</i>						
Total number.....	10	11.	7	17.5	17	13.
<i>Cough.</i>						
Total number.....	88	100.	38	95.	126	98.5
Slight	30	34.	8	20.	38	29.5
Moderate	27	30.	13	32.5	40	32.
Excessive	31	36.	17	42.5	48	38.
<i>Tobacco.</i>						
Total number.....	68	77.5				
Slight	16	18.2				
Moderate	28	32.				
Excessive	24	27.3				
<i>Alcohol.</i>						
Total number.....	37	42.				
Slight	17	19.				
Moderate	14	16.				
Excessive	6	7.				

LXIII.

THE INFLUENCE OF THE NOSE ON EYE AFFECTIONS AS EVIDENCED BY A CASE OF BILATERAL BLINDNESS AND ONE OF UNILATERAL SCINTILLATING SCOTOMA CURED BY OPERATIONS ON THE ETHMOID CELLS.*

BY H. W. LOEB, M. D.,

ST. LOUIS.

Although many reports have been made showing the influence of the accessory cavities of the nose in the production of serious ocular disturbance, we are still in the dark as to the manner by which the condition results.

Furthermore, we do not know why it is that the affection occurs in one instance, when in what appears to the observer an identical or similar condition, nothing of such a nature takes place. We have already been made familiar with those cases of extensive caries of the body of the sphenoid and of the lateral mass of the ethmoid without the slightest disturbance of ocular function. And yet there are other cases in which the accessory cavities are only slightly affected, while the accompanying condition of the eye, be it causal or coincidental, is extremely grave.

The studies in the anatomy of the region made during the past few years have fairly well established the anatomic relations. The pathologic side, however, is still far from satisfactory by reason of the infrequency of autopsies in these cases and the length of time which usually intervenes between the appearance of the symptoms and the performance of the autopsy.

The clinical side has been greatly increased by the numerous additions to the record of the reported cases. To this record I desire to add two, one of blindness and one of scintillating scotoma, both cured by operation on the ethmoid cells.

*Read before the American Laryngological Association, May, 1914.

Case 1.—Bilateral blindness, acute ethmoiditis, entirely relieved by exenteration of the ethmoid cells.—S. P. C., aged sixteen years, unusually large and well developed for his age, was referred to me by Drs. M. H. Post, M. A. Bliss and J. F. Harrison, August 1, 1912. During the previous five weeks he had suffered from great loss of sleep and intense headache, the pain being localized over the supraorbital region on both sides. He began to notice reduction in his vision three weeks before. This increased day by day until, when I saw him, he was almost blind. A few days before, he had a severe bleeding from the nose, resulting from a fall due to his decreased vision. The patient did not complain of any dizziness, though there had been some nausea but no vomiting.

Examination revealed extreme sensitiveness in the region of the inner canthus, above and below. Both inferior turbinates were of usual size, no swelling being manifest. The septum was quite straight, no ridges or deviations being present. Both middle turbinates were greatly swollen, but no pus was at first discoverable. Upon thoroughly cocainizing the middle turbinate area, a thin streak of pus was found coming from the region of the orifice of the posterior ethmoid cells on both sides.

In view of the history and findings, I gave the opinion that his condition was due to an acute ethmoiditis, which, so far as I could then ascertain, was suppurative in character. A radiograph showed the region of both ethmoid labyrinths obscured, frontal sinuses small but clear, and no evidence of trouble in the maxillary sinuses.

Ophthalmoscopic examination at this time by Dr. Post and Dr. Shahan showed vision 1/192 O. D.; 3/120 O. S.; swelling of the disc + 4 D., O. D.; + 6 D., O. S.

I removed a large portion of the middle turbinates and cleared out the anterior and posterior ethmoid cells on both sides, and found the mucosa over the turbinates and within the cells considerably thickened. One cell wall was completely filled with the thickened mucosa which partook of the nature of a granulation. A very small amount of pus was encountered during the operation. The whole appearance was that of a recent marked ethmoiditis just becoming purulent in character.

The patient began to improve immediately. He was prac-

tically blind before the operation, and within twelve hours afterwards he could recognize the large monogram on the hospital spoons. He left the hospital in one week with his vision almost completely restored.

Examination by Drs. Post and Shahan showed progressive improvement in vision as follows:

	O. D.	O. S.
August 1st (on admission).....	1/192	3/120
August 3rd (after operation).....	3/75	3/12
August 4th.....	10/38	10/19
August 5th.....	10/24	10/19
August 6th.....	10/15	10/15
August 8th.....	20/24	20/24
August 29th.....	20/12	20/19
September 18th.....	20/15	20/15
December 14th.....	20/12	20/12

The swelling of the disc, as ascertained by both Dr. Post and Dr. Shahan, showed progressive recession, from O. D. 4 D., O. S. 6 D. on August 1st, to O. D. 1 D., O. S. 1 D. on August 29th.

Since this time he has had no further trouble with vision or headache. It seems to me that we have in this case a condition analogous to that of edema glottidis, supervening upon an adjacent grave infection, as, for instance, in deep suppurations of the mouth and pharynx. In these cases as soon as the focus is relieved the edema rapidly disappears. Here, too, the condition rapidly improved when presumably the infecting tissues were removed.

The literature shows a fairly large number of cases of blindness which has been relieved by surgical attention directed to the accessory sinuses of the nose. While in a few instances the infective focus was in the ethmoid, maxillary or frontal, in the main the sphenoid was at fault. Few cases, however, have shown such a positive dependence on a process in the ethmoid, or such a coincident and consistent recovery as the one here reported.

Case 2.—Unilateral scintillating scotoma; chronic ethmoiditis. Entirely relieved by exenteration of the ethmoid cells.—Miss L. L., aged sixteen years, August 27, 1913, had been having for the past two years daily attacks of severe left-sided

headache, with what she described as flashes appearing in her left eye. These attacks lasted for about five minutes, coming on without any apparent regularity during the day, and without any cause ascertainable on the part of the patient. Furthermore, they had not changed whatever in severity. Very naturally the young lady was very nervous and more or less incapacitated on account of the attacks. She was under the care of Dr. Post for some time, but treatment directed to her eyes and her general condition was without avail. In the hope that something might be found in the nose to account for the symptoms, she was referred to me. She presented no special nasal symptoms except that of sneezing, which she stated had been noted since she was a small child. She had had no headaches except as already specified. Family and other personal history good.

Examination of right nasal cavity showed nothing abnormal except some enlargement of the middle turbinate. On the left side there was a marked hypertrophy of the middle turbinate, the inferior being normal, and the septum not deviated.

There was evidently a chronic ethmoiditis on the left side. Since this corresponded to the side on which the symptoms were declared, it was decided to remove the anterior end of the middle turbinate and to curette the ethmoid cells, although no promise of relief was given. The process was more extensive than I had foreseen, thickened mucous membrane without pus being found in most of the cells, and the anterior end of the middle turbinate being virtually an ethmoid cell of the type of a concha bullosa. An extensive exenteration was therefore undertaken. The patient suffered from a very mild attack on the day of the operation, but since that time, now nine months, she has not had a single attack.

So far as I have been able to find, no report has been made of a case of scintillating scotoma relieved by operation on the nose. In fact, there seems to be no disposition on the part of the writers to associate it with disease of the accessory sinuses. Wilbrand and Saenger,¹ who have written extensively upon the various forms of scintillating scotoma, say that little is known of its origin. They quote the following as to its etiology: "Moebius claims that in ninety per cent of the cases a neuropathic disposition is demonstrable. According to L. Mayer and Ricchi, the attacks frequently occur at men-

struation, and according to Strehl, the condition is brought about by puberty in one who has an hereditary predisposition. Antonelli holds that there is a relation between scintillating scotoma and neurasthenia, hysteria, epilepsy, tabes and progressive paralysis. Most authors look upon the condition as a form of migraine, and ascribe to each the same etiology."

In my case it might be claimed that the operation was no more effective than any other form of operative or suggestive treatment. Admitting such a possibility, we must still take into account the extensive pathologic condition present on that side of the nose, and absence of any promise of relief at the time of the operation.

At any rate, attention to the accessory sinuses in any similar condition will do no harm, and may meet with an equally satisfactory result.

Finally, these two cases bring again into our field of study the observation which was made in my paper on "A Study of the Anatomic Relation of the Optic Nerve to the Accessory Cavities of the Nose,"² that under ordinary circumstances the optic nerve is in close relation with the ethmoid labyrinth only at the posteroexternal angle of the last posterior ethmoid cell. Where this relation exists, there is only the slightest possibility of any danger to the optic nerve in suppuration confined to the ethmoid cells. But when the last posterior ethmoid cell replaces the sphenoid, the optic nerve runs close to and along the external wall of this ethmoid cell (as in two out of thirty specimens studied), and the vulnerability of the nerve is correspondingly heightened, in view of the greatly increased portion exposed.

Without being able to demonstrate it, I feel that this is the relation of the posterior ethmoid cell to the optic nerve in the case of blindness reported, confirmed by the history and course of the disease and the rapid restoration of vision following exenteration of the ethmoid cells.

REFERENCES.

1. Wilbrand and Saenger: *Die Neurologie des Auges*, Vol. 3, part 2, 1906.
2. Loeb, H. W.: *Annals of Otology, Rhinology and Laryngology*, June, 1909.

LXIV.

THE CLINICAL SIGNIFICANCE OF BACTEREMIA.*

By J. E. SHEPPARD, M. D., F. A. C. S.,

BROOKLYN.

Having been asked by our secretary to write upon this subject, I feel like confessing that the title has a distinctly more pretentious sound than the paper warrants. My efforts will be limited to a report of four cases, which have seemed to be fairly illustrative of a considerable series of cases met with by my associate, Dr. Stickle, and myself in our work at the Jewish Hospital of Brooklyn, together with a few deductions drawn therefrom.

Case 1.—Rosalie M., seven years old, was admitted to the Jewish Hospital, August 27, 1913. She had an attack of measles late in the preceding June, followed by an acute suppurating right ear, and on July 17th I opened the mastoid, finding nothing particularly out of the ordinary. No unusual symptoms developed until about August 12th, when, with chilly sensations and vomiting, the temperature rose to 104° , and some fetid pus was found blocked up in the tympanic cavity and antrum. These symptoms continued with more or less severity until her admission to the hospital on August 27th, on which day Dr. Stickle, in my absence from the city, did a further operation. The sinus was opened with resultant free bleeding, no thrombosis being found.

The sinus blood was cultured, no growth occurring until two days later, when there were numerous colonies of the streptococcus longus. This was followed by a period of daily chilliness, fever and sweats, lasting until September 22d, after which the temperature remained flat and further recovery was uneventful. From a culture taken September 13th the blood was reported sterile.

*Read before the American Laryngological, Rhinological and Otological Society, Atlantic City, June, 1914.

Repeated blood counts showed the following results:

	Leucocytes.	Polymorpho- nuclears.
August 27th.....	10,400	86%
August 29th.....	14,000	74%
September 4th.....	22,000	80%
September 9th.....	20,000	89%
September 12th.....	12,000	66%
September 15th.....	10,000	65%

Repeated urine examinations gave negative results throughout. For a considerable period she was given five grains of urotropin every four hours.

On September 1st and 2d she was given a mixed streptococcus vaccine. On the 9th, 13th, 16th and 19th she was given an autogenous vaccine.

At the time of the second operation the child presented a strongly septic appearance and there seemed no doubt about the need of opening the lateral sinus. Although no thrombus was found, it seems more than probable that the blood infection occurred at this point.

Case 2.—H. L., aged forty-four years, male, was admitted to the Jewish Hospital, November 2, 1910, when he had given the following history: Four months ago, with a severe head cold, he developed a severe headache, the pain later localizing in the left ear, but was gone within twenty-four hours, a troublesome tinnitus remaining. The earache returned at intervals, there having been a particularly severe attack just before he came to the hospital.

Examination on the day of admission, November 27th, showed receding acute otitis media, without perforation; there was pus coming from the left frontal sinus, which, however, was not tender even on deep pressure, nor had he complained of pain in that region; there was no pain in the ear and no mastoid tenderness.

Temperature on admission was 104°. On November 4th and 7th Widal reaction was negative. A blood culture taken on the 6th showed streptococcus mucosus capsulatus; another on the 12th the same, twenty-four colonies to the cubic centimeter; November 14th the same, 142 colonies to the cubic centimeter; November 18th the same. On this date the

agglutination reaction, in a dilution of 1 to 800, was strongly positive; in one of 1 to 16,000, was negative. November 24th another blood culture, ten cubic centimeters of blood distributed among five plates and two flasks, was sterile.

Blood counts showed the following:

	Leucocytes.	Polymorpho- nuclears.
November 2d	13,800	74%
November 3d	13,000	85%
November 4th	11,000	83%
November 16th	17,800	77%
November 17th	14,000	73%
November 18th	16,000	76%
November 19th	13,000	70%
November 20th	9,600	65%
November 21st	10,600	75%
November 22d	7,000	54%
November 23d	6,000	73%
November 24th	9,200	77%

The urine examinations were negative throughout.

The patient was not operated, and received no vaccine treatment. He left the hospital, contrary to advice, November 25th, but was obliged to return within the next two or three weeks with two or three metastases, not serious clinically. The same organism, the streptococcus mucosus capsulatus, was found in all metastases.

Case 3.—Samuel Z., aged seven years, was admitted to the Jewish Hospital, April 27, 1914. The present illness began April 11th with headache and pain in the left ear, and temperature of 106°. No cough, no vomiting, no convulsions. There was not at that time, nor has there been since, any discharge from the left ear. Under treatment the temperature gradually subsided, rising to a high degree again on the eighth day, when there was slight rigidity of the neck, slight Kernig, and twitching of the eyes. On April 21st he was seen at the dispensary of the Jewish Hospital, when there was again a high temperature; the left drum membrane was congested; the cervical glands on the left side were enlarged. The temperature subsided under treatment, but was high again on the 24th, since which time there have been chills every afternoon,

followed by high temperature. Widal negative. On admission, April 27th, temperature was 99.8°.

Examination of ears by Dr. Stickle showed right membrana tympani normal; left membrana tympani almost subsided acute otitis media (nonsuppurative); two or three very slightly enlarged glands along the anterior border of the sternomastoid muscle; no mastoid tenderness.

Blood examinations showed the following:

	Leucocytes.	Polymorpho- nuclears.
April 27th	15,000	82%
April 29th	32,600	80%
May 3d	18,000	87%

April 28th: Blood culture sterile.

April 29th: Cerebrospinal fluid, 25 cc. under moderate pressure; clear, numerous fine flocculi; 1 mm. albumin ring; Fehling's reduced; globulin negative. Cytology: A few lymphocytes.

May 1st: Eye grounds negative.

May 2d: The medical side having excluded all other probable causes of high temperature, requested an exploration of the lateral sinus. Operation was undertaken by Dr. Stickle.

A small antrum was found, containing a small amount of coffee colored mucus (not even mucopus) and no granulations; the bony walls of the antrum were intact throughout; the floor of the antrum consisted of the wall covering the knee and adjacent portions of the sigmoid sinus. The latter was exposed from where it turns inward towards the bulb to a considerable distance back of the knee, and was not pulsating. Upon opening the sinus there was found a large thrombus, broken down and containing pus centrally, and extending from the torcular to the bulb. From the latter direction only a slight return flow was obtained, while it was well washed out from the torcular end. The pus from the thrombus showed streptococcus longus. Culture of blood, taken immediately after operation, developed a hemolytic streptococcus, six colonies to the cubic centimeter.

May 6th: Blood culture showed streptococcus longus, three colonies to the one-half cubic centimeter.

May 25th: Blood culture sterile.

Temperature practically flat from May 9th, and recovery

uneventful. No sera or vaccines used. Urine negative upon repeated examinations.

Case 4.—Edward J., aged four years, was admitted to the Jewish Hospital, April 27, 1914. Present illness began four weeks ago with an attack of grippe, with a resultant acute otitis, for which the right drum membrane was incised three weeks ago at the Postgraduate School in New York City, followed by hot irrigations.

There was a low grade temperature until four days ago, when he had a chill, and since then high fever continuing, but fluctuating; was very restless, had headache. Three days ago developed pain in right leg; not localized, but whole leg seems tender to the touch, and he does not move it. There were no pulmonary symptoms, no urinary trouble, but he was very constipated.

On admission, examination by Dr. Stickle showed both drum membranes normal, and no mastoid tenderness. Temperature was 101.4°.

On April 30th in right drum membrane a small hemorrhagic area appeared, but no evidence of inflammation. Same day eye grounds negative.

April 28th and May 1st X-ray showed no trouble with the bones around the hip joint.

Urine examinations were as follows:

April 28th: Negative.

May 13th: A trace of albumin; loaded with white blood cells and bladder epithelium.

May 29th: Faint trace of albumin; no white blood cells or epithelium.

Blood examinations showed the following results:

	Leucocytes.	Polymorpho- nuclears.
April 28th.....	20,000	91%
April 29th.....	17,000	77%
May 9th	8,600	66%

Blood cultures gave the following results:

April 28th: *Streptococcus brevis*.

May 1st: *Streptococcus brevis*.

May 6th: Sterile.

May 12th: Culture sterile. Same date, cerebrospinal fluid,

slightly bloody specimen; 2 mm. albumin ring; Fehling's reduced; globulin negative; blood cells; bacteriology, negative.

May 12th: Drum membranes negative. Recovery from then on uneventful.

These two cases (3 and 4) entered the hospital the same day; neither of them had on admission sufficient ear symptoms to appear specially significant.

Case 3, the operated case, did not have a bacteremia until the day following his operation. Case 4, the case that was not operated, had a bacteremia on admission.

Case 3 on admission had a leucocytosis of 15,000, with a polymorphonuclear count of 82 per cent, going up in six days to 18,000 and 87 per cent.

Case 4 on admission had 20,000 leucocytes and 91 per cent polymorphonuclears; going down in three days to 17,000 and 77 per cent; and in eleven days to 8,600 and 66 per cent.

Case 3 on admission appeared gravely septic. Case 4 did not to nearly as great an extent.

Finally, case 3 needed operation, case 4 evidently did not.

In closing, these cases and others of the group of which these are a part would appear to demonstrate that not all cases of otitic bacteremia need operation.

To distinguish between those cases that do and those that do not require operating would seem to call for our closest observation and best judgment.

As aids to us, often all too meager, it is true, in coming to a conclusion whether or not to operate, I would suggest the following:

1. The general condition and appearance of the patient. Is there a markedly septic condition? Is it increasing or decreasing? And, as of course the greatest aid in determining this,
2. The temperature curve.
3. Whether the process is localizing or tending to become general (if metastases are occurring, etc.).
4. Blood counts frequent enough to keep a careful line on the patient's resisting power.
5. Blood cultures sufficiently often to have a definite knowledge of the persistence of the organism in the blood, and whether the number of colonies per cubic centimeter is increasing or diminishing; thus showing whether or not the patient is in need of assistance in taking care of the bacteremia.

I cannot close without an acknowledgment of my obligation to my associate, Dr. Stickle, who did much of the work, both clinical and other, in connection with these cases, and to our Jewish Hospital pathologist, Dr. Simon Blatteis, whose superior knowledge and judgment of certain phases of most all of the cases has always been at my disposal.

LXV.

A CASE OF SEPTIC INFECTION OF THE PAROTID
GLANDS RESULTING FATALLY.*

By F. E. HOPKINS, M. D.,

SPRINGFIELD.

Lesions about the mouth are so often brought to the attention of the laryngologist, that a brief report of what must be a rare case of this character is worthy of record. The parotid glands, because of their size, importance and anatomic relation to nerves and large blood vessels, afford possibilities of serious complications, and a case of infection of these glands is the subject of this report.

A review of some of these relations to other structures makes for a clearer understanding of the results which may follow serious lesions of the parotid. The gland has three large lobes. The third, or most deeply placed, passes behind the styloid process, and beneath the mastoid process and sternomastoid muscle, coming in contact with the internal jugular vein and the internal carotid artery. From the relation which this carotid lobe holds to the internal jugular vein, it follows that swelling of the gland may cause passive congestion of the brain by compression of that vein. The following structures are found to traverse or to be in intimate relation with the gland: The facial nerve, the temporomaxillary, superficial temporal, internal maxillary and posterior auricular veins; the commencement of the external jugular vein; the external carotid artery which supplies branches to the gland and divides into its two terminal branches—the temporal and internal maxillary arteries; and the terminal part of the great auricular nerve. The posterior auricular branch of the external carotid artery and the transverse facial branch of the temporal artery arise within the substance of the gland. The gland is separated from large vessels and from the pneumogastric, glossopharyngeal and hypoglossal nerves by a thin layer of fascia.

The quotation from anatomic authority illustrates the difficulty of complete removal of the gland, or even of thorough incision to secure complete drainage in case of septic infection. The structure of the salivary glands, too, explains something of the difficulty met with in an attempt to drain by incision, in case of closure of the intralobular ducts, for the salivary glands are of the compound tubular variety, made up of lobules, each lobule consisting of the branchings of a subdivision of the main duct.

Now, in a case of involvement of the entire gland, if we consider each of the great number of the intralobular ducts as occluded by an exudate, the problem of securing perfect drainage is seen to be a difficult one.

Through the courtesy of Dr. Goodell of Springfield, who was about to leave for his summer vacation, I saw on July 1, 1913, B., male, fifty-four years old, who was suffering from an infection of both parotid glands. The cause, or why the development should start as a symmetrical one, is not clear, and was probably due to some unusual sepsis.

There was but slight rise of temperature and little acceleration of the pulse. Though worn to the point of exhaustion by heavy business cares, he kept himself at work, complaining principally of symptoms due to obstruction of Steno's duct, of the great, unsightly swellings at the angles of the jaws, and of mental heaviness and depression. Dr. Goodell had succeeded in probing the duct of the left parotid, and this gland was less swollen than the right, the duct of which had not been entered.

After several trials I succeeded in passing a probe through the right duct down to the gland, and a few drops of thick mucopus drained away, but the inflammatory process in alveoli and ducts had evidently continued too long to permit of adequate drainage through the duct. Imperfect as was the drainage gained by probing, the slight easing of pressure brought a measure of relief. The patient was prevailed upon to drop his business and enter a hospital; and though there were intervals of temporary improvement corresponding to the measure of drainage secured, on the whole the case did badly. The temperature rose to 102° at night with morning remissions, but this mild curve did not correspond to the gravity of the process going on.

On July 18th, Dr. Dudley Carleton, a surgeon, saw the case in consultation with the family physician and myself, and in an effort for better drainage made deep incisions into both glands. From the tense tumor a free flow of fluid might have been expected; but there was only a moderate discharge of thick mucopus exuding from those lobules entered and no diminution of the general swelling. These incisions proved as ineffective as a means of drainage as had probing the ducts. Why these limited incisions failed is seen by recalling the anatomy of the gland and the pathology of the process taking place—the many lobules opened were, in fact, but a small fraction of the entire gland, and the great majority of the lobules still remained undrained because of the closure of their ducts.

Some of the patient's symptoms, particularly the mental heaviness and depression, seemed to confirm the suggestion presented in the anatomic relation of the gland to the internal jugular vein, the great swelling of the gland compressing this vessel and causing cerebral congestion. The mental symptoms certainly were not due to sepsis.

The patient died of exhaustion July 30th.

If a lesson as to the care of septic infection of the parotid gland is to be drawn from this case, the first suggestion is for early attempts to drain by dilatation of the duct, together with efforts to reduce the inflammation of the gland. If such care can begin before the intralobular ducts are closed, the outcome should be favorable. This failing, the glands may be dissected out by a skillful surgeon; and while facial paralysis and much inconvenience from loss of the secretion of the gland must result, the best interests of the patient will be conserved.

LXVI.

TWO CASES OF EXTENSIVE FIBROMYXOMA OF
THE NASOPHARYNX.*

BY JOHN EDWIN BROWN, M. D.,

COLUMBUS.

Few of the various types of tumor of the nasal passages or nasopharynx are so rare as to make their cases of interest merely because of the pathologic group to which they belong. But the location, extent, relation to surrounding tissues and secondary attachments, may make any case of interest from the surgical questions it puts to the operator. These cases are reported because of the surgical difficulties they presented to me.

Case 1.—Male, age seventeen years. The family history was negative on the father's side. The mother, age thirty-five years, suffered from an exophthalmos of the left eye, associated with unilateral choked disc and a central absolute scotoma. A sister of the mother gave symptoms of inherited specific disease. The mother was under observation, taking large doses of potassium iodid, when the son was first brought to me. He was sent in by his family physician, Dr. C. A. C., prepared for a tonsil-adenoid operation which the local conditions plainly indicated. The operation revealed, in addition, an extreme deviation of the septum to the right, which was corrected by operation two weeks later. His right nasal stenosis was thought to be due to this until manipulations during operation revealed a mass projecting from lateral wall of posterior nares apparently directly back of the middle turbinate. It was dense in consistency and gave no free or pedunculated portion for easy surgical attack. The snare was tried unsuccessfully then and on two subsequent occasions, though small masses were secured for the pathologist. March 26, 1908, the inferior turbinate was removed to give better access to the growth. April 8th, under local anesthesia, a loop of No. 5 wire was passed through the nasal passage,

attached to a snare with an extra heavy canula so that the loop encircled that portion of the tumor on the wall of the nasopharynx. The forefinger of one hand was used to keep the wire pressed as closely as possible to the wall of the pharynx as the loop was tightened. It was my original intention to snare off as much of the growth as the loop would engage, and then similarly attack the remaining portion of the growth. As the wire was drawn home it was realized how firmly it was imbedded in the tissue. I decided, therefore, to make traction on the loop and use my finger in the nasopharynx to act as a blunt dissector to aid in separating the tumor from its attachments. The reward was a separation of the tumor in its entirety from its attachments, and its withdrawal—somewhat difficult—through the anterior naris. The only laceration of the surrounding tissues was of a small strip of ethmoidal mucosa which came out with the tumor. The tumor itself was oblong, fifty-five millimeters in length, and twenty-two millimeters in width. Its consistency was that of an ordinary fibroma. The hemorrhage after removal was slight, and there was no surgical shock. The patient has remained entirely well since its removal (five years ago).

Case 2.—G. J. H., age eighteen years. Family history good; father and mother, both paternal and maternal grandparents, all living and healthy. He was referred to me by Drs. Crotti and McKenzie. He had the facial appearance of tonsil-adenoid disease, and had undergone, at the hands of a competent rhinologist, an operation for removal of tonsils and adenoids, and later a second operation under local anesthesia for a condition diagnosed as "polypus." Subsequent to the first operative procedure a slight swelling that had appeared in the right cheek had increased in size, and coincident with this, obstruction to the passage of air through the right nostril had become marked until at the time of his visit it was possible for the patient, by closing the opposite side, to aspirate only a trifle of air through the nostril, while none whatever could be forced outward. Only a short time before, a surgeon of his home town had attempted an external operation on the swelling in the cheek. The operation was discontinued without removal of any tissue, on account of hemorrhage and poor condition of the patient.

Examination disclosed a tumor mass which occupied the

posterior third of right nostril, was confluent with its outer wall, and had become adherent to septum where in contact with this posteriorly. A probe could be passed along the floor of the nostril into the nasopharynx. By posterior rhinoscopy this mass could be seen high in the vault and well to the side, affording no projecting or pendulous portion. The swelling in the cheek was evidently due to the growth outward through the sphenomaxillary fossa of a tumor, presumably the same as occupied the posterior nares. The boy's condition was rapidly growing worse. His physician stated that he seriously collapsed in the recent operative procedures. It was deemed unwise to attempt anything that did not promise the entire removal of the growth, and I advised approaching the tumor through the canine fossa and maxillary antrum. Under ether this operation was begun. The antral cavity was free from growth, but its posterior bony wall had disappeared, and in this region was a dense mass continuous with the tumor in posterior nares and apparently with the mass in the cheek. It was difficult by dull dissection to free the mass from its attachments so that it could be grasped by strong forceps, and in the process a number of smaller masses were torn away from the tumor. The postnasal space had been plugged from below, but the hemorrhage was free and the patient's pulse showed profound collapse so that ether was withdrawn and oxygen alone used during the latter part of operation. The tumor was finally evulsed. It apparently had occupied the space of the posterior ethmoidal cells, sphenoid sinus, the sphenomaxillary fossa, and to a limited extent encroached on the space of the maxillary antrum. After packing, the patient was in serious collapse and pulse was not perceptible at the wrist. Dr. Crotti made transfusion of seventy cubic centimeters of blood from the father to the patient by the indirect method he usually follows. Oxygen was used, for twenty-four hours, together with a large amount of strychnin and atropin. At the end of this time he showed signs of rallying, and from that went on to recovery. At this time he is in the best health he has had for several years, and examination shows no evidence of a return of growth. The region occupied by the tumor has healed, leaving, however, the sphenoid, posterior ethmoid, and maxillary cavities confluent with the nasal passage.

The pathologist's report stated that this tumor was identical in nature with the first—namely, a fibroma of myxomatous character. Some uncertainty had attended the report on the sections from small specimens removed in the first case, but when one was obtained that was definitely from the tumor there was no doubt as to its benign character. Dr. J. J. Coons of Columbus made the pathologic report, confirmed in the first case by Dr. Bloodgood of Baltimore.

[Note.—In December, 1913, case number 2 reappeared with the history of having had several severe hemorrhages. Examination revealed a soft elastic mass in the region of the face of the sphenoid and the adjacent lateral wall of the nasopharynx, from which there was a pulsating flow on puncture with needle. Local anesthesia was applied, and after preparations for checking hemorrhage were made, the snare was used to remove a small mass for examination. The wire cut through the tissue without resistance and a profuse hemorrhage ensued. Before the packing, which was at hand, could be inserted, probably one and one-half pints of blood had been lost. A specimen was not secured. Later attempts at removal of packing during the next four weeks were always followed by profuse hemorrhage. January 1st the common carotid on that side was ligated, but without effect. The packing was then left in place and removed only when indications of decomposition appeared, the bottommost part being never entirely removed until the latter part of March, when but slight oozing took place. At the last dressing there was no hemorrhage. The patient has not been seen now for one month. There is no indication of malignancy, either locally or by metastasis.]

LXVII.

A CASE OF PAPILLOMA OF THE LARYNX
TREATED BY RADIUM.*

By F. E. HOPKINS, M. D.,

SPRINGFIELD.

The subject of papilloma of the larynx in children was thoroughly discussed by this association in 1905. Dr. Clark's exhaustive paper grew out of a large experience with these most serious cases, and his conclusions furnish a prudent and intelligent basis for the care of this disease in children. Notwithstanding the peril to life from suffocation, and the tendency to rapid recurrence after removal, the natural history of the development of these growths is such that a measurably favorable prognosis is warranted. This favorable element is due to the fact that when the period of the child's active growth has passed, the papilloma may disappear spontaneously, or no longer recur after thorough removal; and Dr. Clark states briefly and positively that the best method of treatment is tracheotomy and noninterference with the growth.

Now, the case of the adult suffering from papilloma of the larynx may be far more desperate than that of the child, for he can look forward to no period of cessation of activity in the development of these cells, and rapid recurrence may be the rule. The grim, persistent and unchanging course sometimes run is illustrated in a case reported by Dr. Robert Abbe in the *Medical Record* of April 13, 1912. This case had been under the care of Dr. W. L. Culbert of New York, who with cutting forceps had repeatedly cleared the woman's larynx of masses of papillomata. This case was a legacy to him from Dr. Lincoln, who had done the same, and who only continued to do what had been done by Dr. Elsberg—surely this patient was wise in her choice of laryngologists. The picture of this larynx stuffed with warty growths was published by Dr. Elsberg in his report of the case appearing in the transactions of the American Medical Association for 1865.

Bringing the history of the case down to 1912, as noted above, the patient's larynx had been cleared out every six months for over forty-seven years—a comment on the tendency, or lack of it, of these warty growths to take on malignancy when repeatedly operated upon.

A case similar in character, though less severe in degree, is that of Mrs. C., fifty years of age, who recently came under my observation through the courtesy of Dr. J. L. Barton of New York. Mrs. C. was operated upon for the removal of papilloma of the larynx seventeen years ago. Tracheotomy was done at that time and an ounce of papillomatous tissue removed. She has been operated upon repeatedly since, and the snare, forceps, curette, actual cautery and monochloroacetic acid have all been used. Tracheotomy has been performed seven times during this period of seventeen years.

These obstinate cases are referred to as illustrating the serious problem that some of them present, and because both of these have recently been treated by radium. Mrs. C.'s treatment was by Dr. Abbe on December 23, 1913. Dr. Abbe also treated the first named case. A tracheotomy was done under ether and radium, applied directly to the larynx for a period of forty-five minutes. The growths disappeared and the tracheotomy wound was allowed to close at once, and for a time breathing through the larynx went on reasonably well. Narrowing, however, occurred, and to such a degree that tracheotomy again became necessary and was done April 5th. At this time no growths could be seen in the larynx, its narrowing being due to adhesions through the anterior three-fourths. In fact, the opening remaining was so small it seemed incredible that sufficient air to sustain life could pass. An explanation of this cicatricial contraction which has been offered is that former treatment by acids, cautery, etc., is responsible. At this date a narrow ring of grayish growths can be seen on the under side of the opening in the larynx—a recurrence of the papillomata—and the adhesions within the larynx may be due to burns by radium. If this latter is chargeable to radium, then caution must be exercised in its use, and the recurrence shows the need of repeated applications.

Those who have used radium are claiming that a cure of papillomata of the larynx can be effected by its use. If this

happy result may be reached, and by a method which does not injure the larynx, a boon will be conferred alike on patient and on surgeon. I offer this brief note, hoping your discussion may help to make clear the possibilities of the new agent. Is it to bring about a cure, or, like the uncertainty of surgery, give but temporary relief; and is it also capable of doing damage to the larynx itself by burning and subsequent cicatricial contraction?

SOCIETY PROCEEDINGS.

TRANSACTIONS OF THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY.

MEETING AT ATLANTIC CITY, JUNE 19 AND 20, 1914.

SYMPOSIUM: STENOSIS OF ESOPHAGUS.

(a) Anatomy, Anomalies, Instruments and Technic.

DR. HARRIS P. MOSHER, Boston, after discussing briefly the comparative anatomy of the esophagus, considered the esophagus in man under the following subheads: (1) Congenital anomalies; anatomy; structure; lymphatics; position; direction; diameter; length; distensibility; subphrenic portion; movements; appearance of normal esophagus. (2) Physiology; function; peristaltic action; respiratory movements; innervation.

He then discussed the contraindications to esophagoscopy. The only contraindications to the performance of esophagoscopy are acute inflammation, as after the swallowing of corrosive fluids, and aneurism of the aorta. The chief danger in passing the esophagoscope is rupture of the esophagus, which almost always results in infection of the posterior mediastinum and death. Such an accident can be easily avoided by the selection of a tube of the proper size and by adhering to the fundamental axiom of all esophageal examinations, namely, the examining tube must never be advanced unless the eye sees through the tube the open esophagus ahead. It should be remembered, also, that in old people the esophageal wall may be thin enough to rupture itself, so that smaller tubes and greater care in using them are necessary. It has developed of late years that there is considerable shock from manipulations carried out in the esophagus, more shock than is caused by working in the trachea and bronchi. Chil-

dren bear esophageal examinations less well than do adults. In poorly nourished patients, particularly those who are on the point of starvation from the presence of a stricture, it is better practice to open the stomach and feed the patient through a gastric fistula until his resistance has been restored, before attempting any prolonged examination.

The esophagus may be examined under local or general anesthesia, the author being strongly prejudiced in favor of general anesthesia, doing most of his esophageal work with ether.

The laryngologist, on beginning esophageal work, should supply himself with a full set of general and special instruments, a full list of which is recommended in the paper.

A general physical examination of the patient should be made before esophagostomy is attempted. An X-ray plate is indispensable before many examinations. It shows the location of metallic foreign bodies, pieces of bone, and buttons; it shows enlargements of the arch of the aorta and enlargement of the mediastinal glands, and, combined with the ingestion of bismuth, it shows the position of strictures, the size and location of diverticula, and the size of the dilated esophagus.

The technic of esophagoscopy under local and under general anesthesia is described in detail.

(b) Causes, Symptoms, Pathology, Diagnosis and Treatment.

DR. CHEVALIER JACKSON, Pittsburg: Stenosis of the esophagus may be classified as inflammatory, neoplastic, compressive, spastic, and angioneurotic.

The inflammatory stenoses may be edematous or cicatricial; the neoplastic, benign or malignant. The compressive may be due to malignant, benign or inflammatory, or periesophageal lesions, or to aneurism or enlargement of the left auricle. Compression stenosis is also frequently caused by a pulsion diverticulum of the esophagus itself when the pouch is full of blood. Spastic stenoses are usually seen at the cricoid level, due to spasmodic contraction of the inferior constrictors, especially the orbicular fibers, along with the adjacent orbicular fibers of the esophageal wall. The next most frequent spastic stenosis is at the hiatus, the contraction being due to the traction of the diaphragmatic musculature and the

small bundles of fibers given off from the diaphragm and attached to the esophagus. Angioneurotic edema producing severe stenosis of the esophagus has been observed by Arrow-smith.

The etiology varies with the character of the stenosis. Inflammatory stenoses are most frequently caused by swallowing corrosives, but they may be due to stasis of food halted by spasmodic stenosis. Other causes are the traumata of foreign bodies, the mixed infections associated with lues and with tuberculosis of the esophageal wall.

The cause of neoplastic stenosis, like that of neoplasms elsewhere, is unknown. Inflammatory processes and their end-results probably contribute to the etiology by affording a favorable soil.

Spastic stenosis may be local or general. Local stenosis is often the result of a "vicious circle" started by rapid eating, with its associated gulping of large boluses of illy masticated food, followed by stasis of the food, inflammation and erosion, which, in turn, serve as a source of reflex excitement of more frequent and prolonged spasm, this inflammatory process being thus increased. The general causes of spastic stenosis are dependent upon some basic nerve disorder, probably more functional than organic.

Pulsion diverticulum, as demonstrated by Killian, is chiefly caused by insufficient support of the esophageal wall between the fundiform and oblique fibers of the inferior constrictor.

Extensive consideration of the symptomatology is useless, inasmuch as any abnormal sensation whatever, referable to the esophagus, is an indication for esophagoscopy, by which, rather than by symptoms, the diagnosis is to be made. An otherwise altogether unaccountable cough, "globus hystericus," and the filling to overflowing of the pyriform sinuses with secretions, are some of the signs which may lead to the diagnosis of esophageal stenosis.

Radiography is of the greatest usefulness in demonstrating the size and extent of dilatation and diverticula, as well as assisting in making the diagnosis of their presence. Fluoroscopy affords very important aid by determining the functional activity of the esophagus during the act of swallowing. Taken alone, however, these means may lead to error.

The bougie, passed blindly, is of no use for diagnostic pur-

poses. The differential diagnosis of lues, tuberculosis, and malignancy is made by exclusion and by biopsy. The other types of stenosis are diagnosticated by characteristic signs, which are detailed by the author.

Treatment should never be instituted while the patient is in the condition of water starvation. Enteroclysis or hypodermoclysis should be begun immediately. Immediate gastrostomy by the general surgeon, preferably under local anesthesia, should be performed when the patient has had little or no water for as much as four days.

Edematous inflammatory stenoses are best treated by the swallowing by the patient of small doses of bismuth with a little calomel, both given dry on the tongue.

Cicatricial stenoses, in the author's experience, are best treated by filiform silk woven bougies passed esophageally.

Benign and endoesophageal growths are probably readily removable, though they are so rare that accurate data are lacking.

Malignant stenoses of the esophagus are regarded by general surgeons as inoperable. There is reason to hope, however, that, when early diagnosis is possible, transthoracic esophageal resection may be a justifiable procedure.

Spasmodic stenoses are best treated by divulsion. For this the author prefers a mechanical divulsor, such as that of Mosher, placed by sight through the esophagoscope.

The treatment of compressive stenoses obviously depends upon the nature of the compressive mass.

Pulsion diverticula, according to the consensus of opinion, steadily increase in size and severity of symptoms, consequently amputation of the sac by the surgeon, operating through the neck, is indicated. The operation with esophagoscopic aid devised by Dr. Otto C. Gaub has been successfully employed by the author in two cases. This method is described and illustrated with three figures.

DISCUSSION.

DR. ROSS HALL SKILLERN, Philadelphia, recommended the Schwebe laryngoscope, which, with the suspension apparatus, gives a technic as simple as the passing of a nasopharyngoscope into the nose. With the tongue depressor and the apparatus for lifting up the larynx, the pyriform sinuses and

the upper part of the larynx could be shown completely, without lifting the cricoid cartilage entirely. The instrument was fitted with a double light. The tube was passed as one would pass the nasopharyngoscope, and slipped down below the cricoid cartilage. By comparing this with the other method he had found it the simpler. He could see directly into the larynx. Children, as a rule, stood the esophagoscope badly, but under the Schwebe or suspension apparatus they would stand for hours, or as long as necessary. He had had no child, thus far, to "go bad" with the Schwebe in position.

DR. SIDNEY YANKAUER, New York City, recounted an experience with a stricture at the cardiac end of the stomach. The patient had suffered for many years from cardiospasm, and many attempts had been made to dilate the cardia, all without the slightest effect upon the cardiospasm. The man was unable to swallow other than liquid food, most of which came back into the mouth. He finally came into the hands of Dr. Willy Meyer, who, in the differential pressure chamber, opened the thorax, found the dilated portion of the esophagus, which he corrected by plicating, but without improvement. The patient was put in the chamber again and an operation similar to Mikulicz's pyloroplasty performed on the cardia through the chest wall. There resulted not the slightest improvement in the swallowing or the cardiospasm. Dr. Meyer then turned the patient over to him to see what could be accomplished with the esophagoscope. Upon putting in the 'scope the scar tissue from the plications could be seen, forming ridges at the anterolateral portion of the cardia; beyond this, a bend of the lumen was located at the cardia, which it was impossible to pass. The lumen of the cardia was merely a slit. Attempts to pass through this brought so much pressure to bear upon the sides of the esophagus that it was impossible to proceed. To pass and straighten out this tissue was a problem. He made for the purpose a wire probe which consisted of small joints, about a quarter of an inch long, so constructed that it would bend in one direction, but could not return beyond a straight line. His idea was to use the curved part to go round the ridge to rotate the instrument and then to pass the straight part into the stomach. After several attempts he finally succeeded in straightening out the ridge and in passing a narrow tube beyond the ridge.

He then saw what seemed to be a string coming out from the lumen of the cardia. It proved to be a silk suture from the cardioplastic operation. Subsequently he found another stitch which he cut and removed. Following the removal of these two stitches the scar tissue softened up considerably, after which the gastroscope could be passed. After this the patient could swallow liquid food without difficulty, and some semisolid food. He then passed the gastroscope and manipulated the head and shoulders in an effort to loosen up the cicatricial tissue. The patient's condition improved to such an extent that he could swallow gruels and semisolid foods, but not solid food.

The case illustrated what could be accomplished by the exercise of patience and a certain amount of resourcefulness.

DR. SAMUEL IGLAUER, Cincinnati, described a procedure employed in the treatment of a case of complete obstruction of the esophagus following typhoid fever, occurring in a boy about ten years of age. A gastrostomy had been performed, through which the patient had been fed for a number of years. In order to measure the thickness of the obstruction an olive tipped bougie was passed from the stomach into the esophagus and another was passed through the mouth and the distance between the two olives was determined by an X-ray picture. The olives were found to be slightly overlapped and about one-sixteenth of an inch apart.

Esophagoscopy, both from the oral and the stomach end of the esophagus, failed to reveal any fistulous tract through the obstruction. A few days later, working in conjunction with Dr. Murphy, of Cincinnati, an attempt was made to relieve the obstruction by an original method. An esophagoscope was introduced through the stomach end of the esophagus and at the same time a second esophagoscope was introduced through the oral end. Both tubes were introduced as far as the obstruction, there being an observer for each instrument. When the light in one instrument was turned out the glow of the light in the other could at times be seen transilluminating the obstruction.

With an electrocautery an attempt was then made to burn through the transilluminated diaphragm from above. The cautery was followed by a slender bougie which apparently passed through, but digital examination showed the bougie

under the mucous membrane in the stomach. The bougie had evidently dissected up the mucosa of the esophagus and passed down into the stomach wall. After a few days the patient was removed from the hospital, contrary to advice. While on the train on his way home he began to swallow naturally, and this continued up to the time of his death, which occurred about three weeks after the operation. Evidently the lumen of the esophagus had been restored, and if the procedure outlined above was followed more cautiously it would prove more successful.

DR. GEORGE F. COTT, Buffalo, added a third condition to the two mentioned by Dr. Mosher in which the ophthalmoscope could not or should not be passed. He cited a case in which the patient could not swallow, and in whom, under ether, the esophagoscope could not be passed, after strenuous efforts for some time. Upon further investigation it was discovered that there was ankylosis of the cervical vertebræ from syphilis.

DR. G. HUDSON MAKUEN, Philadelphia, asked if Coley's fluid (mixed toxins of bacillus prodigiosus and streptococcus erysipelatosus) had been tried in malignant disease of the esophagus. Many cases of marvelous cures have been reported, not only by Coley himself but by other distinguished surgeons in this country and abroad, and it would seem that, unless there were some contraindication against its use, it might prove to be quite as efficient, if not more so, than any of the other methods which have been suggested, not excluding radium. He had had the pleasure recently of talking with Dr. Coley about the preparation of the "fluid," and about its use—a work in which he has been engaged for more than twenty years—and he was encouraged to hope that it might be useful in the class of cases under discussion. He would be glad to hear the opinion of the essayists on the subject.

DR. ROBERT LEVY, Denver, said the sign of which Dr. Jackson had spoken—the accumulation of secretion in the pyri-form sinus—had been observed by him in cases of tuberculosis with painful deglutition. He had seen cases in which these sinuses were completely filled.

DR. R. H. CRAIG, Montreal, suggested the use of the high frequency fulguration spark in the treatment of inoperable

cases of this nature. He cited a case in which the pain absolutely disappeared and the growth was greatly reduced. The patient, however, died six months later. In another case of malignant disease involving the sphenoid and the nasopharynx, after a month's treatment the pain disappeared and the growth was much reduced in size. He believed the high frequency fulguration spark is of great value in alleviating pain and diminishing the growth in these desperate cases. Where the growth was accessible, carbon dioxid snow might be useful.

DR. JOSEPH C. BECK, Chicago, asked Dr. Jackson if he had employed the Abderhalden test for carcinoma. He also asked Dr. Jackson to specify, in closing the discussion, what he meant by "small quantities" of radium. He had employed nineteen milligrams of the salt or ten milligrams of the pure radium element.

DR. MOSHER, in closing the discussion, said Dr. Skillern's remarks interested him. He had tried the procedure of suspension esophagoscopy, and had found it satisfactory in the examination of the upper part of the esophagus. He had not had the trouble with the breathing which Dr. Skillern mentioned; in fact, he had been astonished to see how little trouble he had had in this regard. Referring to Dr. Iglauer's case, he said he had reported a case three or four years ago where the same procedure was used. It was a case of absolute stenosis of the esophagus, and he attacked it from above and below. The procedure which helped was that of picking the stricture apart, ballooning, and picking it apart again. In his experience, perforating the esophagus led to fatal mediastinitis.

DR. JACKSON, in closing the discussion, said Dr. Mosher was the highest authority on the anatomy of the esophagus. Dr. Jackson believed that all the early esophagoscopists and many of today mistook the hiatal constriction for the cardia. The question of anesthesia was a matter of the personal equation. Every man should select the instruments which suit his needs best, and so it is with anesthesia. The method should be selected which suits the operator's needs in the particular case. Personally the speaker had been very much interested in the case reported by Dr. Yankauer. He thought the work of Dr. Yankauer and Dr. Meyer would open a new field.

The procedure mentioned by Dr. Iglaue was exceedingly interesting, and while it was a failure in the case cited, it was justifiable in absolutely impervious strictures, and offered hope of success. In cooperating with Dr. Brenneman he had passed a tube to serve as a staff upon which Dr. Brenneman cut the occluding cicatrix from below through the externally opened stomach. Such procedures were, of course, indicated only in impermeable occlusion. If any lumen existed, however small, it could be cured by the safer endoscopic methods. Dr. Cott's point was well taken. Answering Dr. Goldstein's question concerning recurrence of spasmodic stenosis, the speaker cited a case of spasmodic stenosis, in which he absolutely failed. He stretched the abdominal esophagus so completely that he could see into the stomach, and yet recurrence took place. This was, however, his only failure so far. The patient became discouraged and finally gave up treatment. Referring to Dr. Makuen's remarks concerning Coley's fluid, he said the method was applicable to sarcoma, and that it was in this disease that radium also gave best results, carcinoma being less amenable. Both methods could be used in the same patient. He suggested that in the cases of filling of the pyriform sinuses by secretions, mentioned by Dr. Levy, there might possibly be spasm in the cricopharyngeal muscle, causing temporary stenosis with resultant filling of the pyriform sinuses, or, in other instances, a disinclination to swallow because of the odynophagia which might exist. Or in still other instances, an actual organic esophageal stenosis due to tuberculous infiltration of the "party wall" might be present. Nevertheless, Dr. Levy's point was well taken. Dr. Jackson hoped that fulguration and carbon dioxid snow would be thoroughly tried out, but he had had no experience with either. He had not employed the Abderhalden test, as suggested by Dr. Beck, but all diagnostic methods should be used. With reference to the dosage of radium, he said a small dose, such as ten milligrams, acted as an irritant, because it cannot be left long enough in the esophagus. A hundred milligrams left in for an hour or more was usually required. He did not speak as an authority; he depended upon Dr. W. H. Cameron and Dr. William Proescher for dosage and duration of the radium application in each particular case.

Suspension Laryngoscopy in Children.

DR. ROBERT LEVY, Denver: Since the discovery of the laryngoscope by Garcia no more interesting chapter in the history of laryngology has been written than the most recent one by Killian, in which he describes the discovery of suspension laryngoscopy. Since Killian's original communication, suspension laryngoscopy has been elaborated, its limitations and advantages have been carefully proved and commented upon. It has been generally conceded that in adults its limitations are greater than in children. All observers early recognized its value in children, even before it had been tried out in many cases.

The suspension method possesses many advantages over the ordinary direct endoscopy. These are: The entire field and surrounding parts are clearly before one; both hands are free; the dangers from asphyxia are practically nil; and a remarkably clear view may be obtained of the anterior commissure of the larynx and trachea. The after-effects of the suspension method are of no more serious moment than are those of the most simple laryngeal manipulation.

The technic of the procedure in children differs but slightly from that in adults. It may be said to be somewhat easier, although in no event can this procedure be considered a difficult one. The tongue need not be drawn forward; in fact, little attention need be paid to it except to keep it in the median line. The head need not hang far over the table; in fact, if a pillow be placed under the shoulders, the head may even rest upon the table. In many instances, especially when examining or operating in the esophagus, the spatula need not be brought over the epiglottis, but, resting well at the base of the tongue, it will bring the parts into perfect view.

The question of anesthesia is an important one in children. As a rule, local anesthesia is not entirely satisfactory. On the whole, general anesthesia, preferably with chloroform, has given the author the best results.

The specific uses to which suspension laryngoscopy has been applied are: for examination, for diagnosis, for operation through the nasal passages, for the removal of foreign bodies.

A brief summary of the uses to which suspension laryngoscopy has been put and which have been reported is given, to-

gether with histories of six cases in which the method was employed by the author.

DISCUSSION.

DR. SIDNEY YANKAUER, New York City, agreed with all Dr. Levy said concerning suspension laryngoscopy. He had performed a number of operations with this apparatus, one of which he had reported before the western section of this society. After several operations by the general surgeon, the patient's neck was so scarred and the lumen of the larynx was so small that he could hardly get a probe through. The arytenoids were so adherent to the epiglottis that by the ordinary direct methods the lumen could not be seen, but with suspension laryngoscopy this could be accomplished. The passage of the first probe was followed by a larger, and finally he could pass a uterine dilator. By means of uterine dilator he could stretch the larynx sufficiently to get a rubber tube in, which was left in for two weeks, when a larger one was used. It was thus possible, finally, to introduce the intubation tube. The boy is now going about, and has been wearing the intubation tube for nearly four months. The suspension laryngoscope had been modified by Killian himself as well as by others. The new model which Killian had brought out does not appear as useful as the original model. He had not been able to bring the anterior commissure into view with the new device.

DR. SAMUEL IGLAUER, Cincinnati, was interested in Dr. Levy's remarks, and agreed with practically every point. While it was true that the procedure was less difficult in children, it could be carried out satisfactorily in adults. In bringing the anterior commissure into view one should use counter pressure with Albrecht's instrument or the finger. Killian recommended a dose of codein preliminary to the anesthetic in children. He had frequently followed this suggestion. Atropin should be used with ether in order to dry up the secretions. He had tried tonsil removal under suspension, but thought the Beck method far superior and much simpler than that of Killian. Referring to the removal of a broken safety pin from the larynx of a child five years of age, he really did not completely suspend the patient. He introduced the spatula and while supporting the spatula (and thus the patient's head)

with his left hand, he could see the foreign body and removed it immediately with the forceps in his right hand. He had recently introduced radium into the larynx in the treatment of a papilloma in a young child. In another infant radium was applied to the outside of the larynx. He did not know the dosage because he did not own the radium.

DR. THOMAS J. HARRIS, New York City, confirmed what Dr. Levy had said with reference to the simplicity of the method in children and the wonderful view of the larynx obtained in the average case. He referred briefly to a case in which he applied radium in a child six years of age, using the suspension apparatus, with rectal anesthesia, one hundred milligrams of radium, of one million activity, being applied for twenty minutes. The first time he attempted to apply the radium, without the suspension apparatus, he thought it went into the esophagus instead of the larynx. When the apparatus was employed it was very much easier to see the papilloma and to make the application of radium. He recommended the use of rectal anesthesia in these cases.

DR. LEVY, in closing the discussion, had not found the report of Dr. Yankauer's case in his search for all cases to date in which suspension laryngoscopy had been employed in children. He thought the old Killian model of suspension apparatus better in children, but in adults he liked the Albrecht or Howorth modification. He had used cocain in adults, but in children he used general anesthesia. Morphin and hyoscin in cases in which a large percentage of cocain did not seem to abolish the laryngeal reflex were useful in adults. He had never been able to see why adrenalin was added to the cocain, inasmuch as it is not an anesthetic and as there is very little loss of blood in these cases. In some cases of children with chloroform anesthesia, the adductors contracted, the vocal bands came together, and the spasm of the larynx continued for some time. In such cases a dilute solution of cocain was of advantage.

The Clinical Significance of Bacteremia.*

DR. JOHN E. SHEPPARD, Brooklyn: Four cases are reported, which seemed to the author to be fairly illustrative of a considerable series of cases encountered at the Jewish Hospital

*See page 864.

in Brooklyn. The four cases, and others of the group from which these were selected, would appear to demonstrate that not all cases of otitic bacteremia need operation. To distinguish between cases which do and those which do not require operation calls for one's closest observation and best judgment. As aids, all too meager at times, it is true, in coming to a conclusion as to whether or not to operate, the author suggests the following points: (1) The general condition and appearance of the patient. Is there a markedly septic condition? Is it increasing or decreasing? As of the greatest aid in determining this: (2) The temperature curve. (3) Whether the process is localizing or tending to become general. (4) Blood counts, frequent enough to keep a careful line on the patient's resisting power. (5) Blood cultures, sufficiently often to have a definite knowledge of the persistence of the organism in the blood, and whether the number of colonies per cubic centimeter is increasing or diminishing, thus showing whether or not the patient is in need of assistance in taking care of the bacteremia.

DISCUSSION.

DR. ARTHUR B. DUEL, New York City, thought the interesting reports by Dr. Sheppard served to emphasize certain principles in infectious processes which were of great clinical importance. It must be evident that in all infections the general clinical symptoms usually noted are the result of the action of bacteria or their products which have been poured into the circulation. When the general clinical manifestations are slight, the infecting focus is confined to an unfavorable locality for getting into the circulation, or the invading organism is slightly virulent. Thus, a follicular tonsillitis of streptococcic origin, will produce much more violent manifestations than a streptococcic otitis or mastoiditis. Similarly, a staphylococcic follicular tonsillitis, being in a favorable field for pouring its products into the circulation, may cause considerable general upset (though comparatively less than its more virulent cousins), while, confined to a bony cavity, it may cause hardly enough disturbance to be recognized. Whatever manifestation there may be, however, is undoubtedly caused by the bacterial invasion of the circulation. It may be asked, then, why bacteremia is not always demonstra-

ble in suppurative conditions. The answer is twofold. In the first place, the methods of examination are not sufficiently delicate; in the second place, most of the organisms are destroyed almost immediately by the blood cells. Only a few of the most virulent type are able to live and multiply in the circulation. Is a demonstrated bacteremia, then, of no value as an operative indication? Of course it may be of the greatest value. In a case, for example, in which bacteremia is demonstrated, where the blood count shows a low resistance, and where the patient is doing badly, the obvious thing to do is to localize the process—to cut off the blood stream at that point—without regard to whether or not there is a demonstrable disintegrating clot. In fact, in his opinion, to look upon bacteremia alone in suppurative otitis or mastoiditis, as indicative of septic sinus thrombosis, is unwarranted. The bacteremia always precedes the clot formation—indeed, the alteration of the vessel wall by bacterial invasion is essential to the formation of the clot. At this stage it may not be always demonstrable by present methods, but theoretically it is present, and if methods of investigation were nice enough it might possibly be demonstrated a day before, or perhaps a week before, a recognizable clot has formed. Of course, after the clot has formed and has begun to disintegrate, the chance of demonstrating a bacteremia is much greater, because the infecting focus is in the most favorable situation for the constant discharge of large numbers of organisms into the circulation. Yet, with a patient doing badly, one might feel quite justified in isolating the infecting area, whether bacteremia could or could not be shown. On the other hand, with the patient doing very well, the demonstration of a bacteremia might not necessarily demand an operation. Hundreds, of course, recover every year without its having been thought necessary to demonstrate the bacteremia, which is undoubtedly present, and will doubtless continue to do so. One should carefully avoid taking too narrow a view of the clinical significance of bacteremia.

DR. CHARLES W. RICHARDSON, Washington, considered the demonstration of bacteremia an important point in the history of all cases of probable sinus infection. Whether a positive bacteremia is always indicative of sinus thrombosis was a question still subject to considerable dispute. In cases on

the borderline, as it were, the condition of the blood was of much value to the clinician, and should be of great assistance in deciding whether operative intervention was demanded. Without clear and definite indications from a clinical point of view, there was no doubt that many would be loath to go into the sinus, although the experience of Gruening and others would seem to demonstrate that in cases of simple bacteremia, without very pronounced clinical symptoms, there might be sinus thrombosis. How far one would be warranted, with the evidence of bacteremia and without clinical evidences of sinus thrombosis, in delaying operative intervention was a question. With bacteremia, even without clinical evidence, he would be loath to delay. Dr. Sheppard's cases were almost negative, in a certain sense, especially his nonoperative cases, in which he was fortunate in that they recovered without operative intervention.

DR. SEYMOUR OPPENHEIMER, New York City, was under the impression that Dr. Duel and his confreres at the Manhattan Eye and Ear Hospital were no longer at variance with Dr. Libman and Dr. Celler and others at the Mount Sinai Hospital with reference to this question of bacteremia. At Mount Sinai it was held that acute otitis per se never caused bacteremia, and these views had been corroborated by the clinical findings. This difference of opinion was thought to be largely a matter of variance in laboratory technic, the fact that the Manhattan Eye and Ear Hospital reached different conclusions being considered at Mount Sinai to be due to some error in laboratory technic. He and his associates at the latter institution had not changed their viewpoint at all, being still of the opinion that suppurative otitis per se does not cause bacteremia. They were also of the opinion that demonstrable bacteremia is a clinical expression of sinus thrombosis. Up to the present time they had had from ninety to ninety-five cases of sinus thrombosis in which bacteremia had been demonstrated in advance of operation, and in not one case had the operation failed to demonstrate sinus thrombosis. It was a curious fact that if they were wrong in their deductions, they were able to demonstrate conclusively, a few hours after operation, that the blood culture became sterile, showing that the operative procedure must have attacked some local focus which was responsible for the infection. In two hours, in

some instances, the blood had become sterile. In some cases the blood culture still remained positive, but there were other explanations, such as vegetations on the heart valves secondary to the initial infection.

DR. S. MACCUEEN SMITH, Philadelphia, asked Dr. Oppenheimer whether he depended upon the blood cultures for indications for operation, or whether he was guided by other symptoms of the disease.

DR. WILLIAM B. CHAMBERLIN, Cleveland, said if he understood Dr. Sheppard correctly, he opened the lateral sinus but made no mention of ligating the jugular. He could see no reason, under the circumstances, for not immediately ligating the jugular.

DR. EWING W. DAY, Pittsburg, called attention to one point that seemed to have been overlooked. There was a tendency to take for granted that every lateral sinus patient, unless operated upon, dies. If a patient with a thrombosis got well, it was no indication that the sinus was not blocked. He had had cases in which the sinus was completely obliterated by clot, and he could not say that if the patient got well there was no clot.

DR. OPPENHEIMER, continuing the discussion, said, in answer to Dr. Smith's question, he had been particularly cautious, by reason of these controversies, in not being too enthusiastic. Many cases had been admitted to the hospital with but the fewest of otitic symptoms, with suggestions of typhoid fever, possibly, with no symptoms of mastoiditis, but with a positive blood culture. He had been extremely cautious in saying he was dealing with infectious phlebitis, and had deferred operating until all other possible causes had been ruled out. Invariably they had found a thrombotic condition in the sinus.

Answering a question by Dr. George L. Richards as to whether all his ninety or ninety-five cases were operated upon, he said they were.

DR. WENDELL C. PHILLIPS, New York City, verified Dr. Day's statements. He had been struck, years ago, in operative surgery on the ear, to find occasionally an obliterated sinus from an old sinus thrombosis.

DR. JOHN A. THOMPSON, Cincinnati, recalled having treated a brother laryngologist for suppurative ethmoiditis. He

developed an abscess in the big toe, which was opened under antiseptic precautions, and examination of the pus revealed the presence of streptococci. The nasal accessory sinuses were sometimes at fault in bacteremia.

DR. SHEPPARD, in closing the discussion, said he simply reported four cases as a part of a group of cases of bacteremia, many of the group being cases which had been operated upon. Regarding the tying of the jugular, he had rather settled down to the rule of not tying it if there is a reasonably good return flow from the bulb. Should there be no return flow, he waited a day or two and then tied off the jugular. It was probably true, as Dr. Richardson suggested, that he was fortunate in not having bad results. Possibly more than simple good fortune is indicated by the fact that the cases referred to were selected from perhaps fifty or more cases of bacteremia, of which he had kept careful records, and of which twenty-five at least had not been operated upon. Dr. Thompson referred to other sources of bacteremia. This fact needs always to be kept in mind. He very decidedly does not advocate operating upon all cases of bacteremia. Dr. Oppenheimer spoke of his cases as being cases of suppurative otitis. Three of the cases reported in the paper were nonsuppurative otitis. He questioned the necessity of operating upon all these cases if they are properly watched, especially if in conjunction with a practical bacteriologist. He thought more extensive observations would ultimately teach in which cases to operate and in which surgical intervention was not necessary.

Treatment of Purulent Cerebrospinal Meningitis.

DR. WILLIAM SOHIER BRYANT, New York: The object of this communication was to apply the experience of an unusually successful case to the management of this infection.

The treatment of purulent septic meningitis consists of (1) treatment for relief of the intracranial pressure; (2) treatment of the toxemia; (3) treatment of the focal infection. The goal of the treatment is the control of intracranial pressure and toxemia, and the treatment should, therefore, be symptomatically directed against these two means of fatal terminations of the disease.

The following case, showing recovery from purulent streptococcic cerebrospinal meningitis, was offered: The patient,

male, twenty-two years of age, with symptoms of rapidly increasing coma, neuromuscular signs of meningitis, rigidity of neck, choked discs, and purulent otitis media and mastoiditis. Temperature, 102.5°; pulse, 40. Spinal fluid contained cocci in pairs and short chains, and pus. Decompression at once removed the mental symptoms. Mastoid operation, hypodermoclysis, enemata, saline solution by mouth, and solution of magnesium sulphate by mouth. On the seventh day following decompression, all signs of meningitis had disappeared. Patient died one hundred and eighty-eight days after the decompression operation, from toxemia caused by the repeated secondary infection of the decompression wound.

From this case the following conclusions are reached: "The combination of our experience as otologists with the experience of the obstetrician, makes the outlook for successful treatment of streptococcic meningitis appear much brighter than it has previously appeared. Otolaryngologists should get as good results in cerebrospinal meningitis as the obstetrician obtains with puerperal sepsis cases. Although the surgeon can readily protect the patient from death by intracranial pressure, the management of the sepsis is quite another problem. This problem of sepsis has received more attention from the obstetrician than from any other medical group or specialty. The treatment should be focused on decompression, local and systemic drainage, administration of magnesium sulphate, and stimulating general hygiene."

The Operative Treatment of Meningitis—Supplementary Report and Analysis of Cases.

DR. SAMUEL J. KOPETZKY, New York: The author reiterates the statement with which Dr. Irving S. Haynes prefaced his argument presented before this society in 1912, advocating the trial of "drainage of the cisterna magna" to cure certain types of purulent meningitis: "Septic conditions are usually surgical opportunities and demand surgical treatment."

The operative treatment of purulent meningitis had undergone scientific inquiry ever since the above statement was made, and the author presented the position now held by both Dr. Haynes and himself, after two years of further study and experience with meningitic infection.

The object of the Haynes operation—the successful evacuation of pus accumulations and the eventual restoration of an unhindered blood supply to the vital medullary centers—and the author's work of disentangling the complex picture of meningitis so as to give data upon which an early diagnosis might be based, thus leading to a very early institution of this surgical relief before the disease had exhausted the patient and general sepsis had supervened, were fulfilled, and yet the establishment of drainage and its accompanying phenomena failed to secure the desired ends.

Critical study of the cases operated on presents the basis for the opinion that meningitis is not simply a surgical problem, to be solved by finding a safe method by which to eliminate the pus accumulation in the cerebrospinal fluid and to establish intracranial decompression; the analogy between the situation presented in purulent involvement of the meninges and infections of the peritoneum, or the pleura, does not hold. The change in the brain tissue is a factor of great moment in the aspect of the situation as at present considered.

In the author's opinion, a larger number of intrameningeal invasions are brought about by the hematogenous route, and in otogenic meningitis the mechanics of infection are analogous to those which are found in the tuberculous and meningococcus types of meningitis.

If this hypothesis be accepted, improvement is not dependent, as has been maintained by some observers, on the lack of toxins generated and washed away from the blood stream, but is due to the remission of symptoms wholly due to decompression effects on the vital centers, and the unembarrassed blood supply to the brain as a whole. The period of improvement really marks no advance toward the cure of the meningitis, but is only an evidence of the control of the pressure factors in the case. The study of the cases leads to the belief that the final stage is not one of sepsis, as has been held by some. These patients, even when they ran high temperature curves, did not present themselves as individuals suffering from sepsis. The blood examinations which were conducted in this stage were uniformly found negative for cultures on various media. The manner of death differs from that of death by sepsis. The patients all die suddenly, without paralytic phenomena, as if some unknown element sud-

denly overwhelmed them. The manner of death is significant of the inflammation of the brain tissue, which is progressively advancing in nature, and when the cells composing the brain itself are affected to a given degree, unknown in its definitiveness by means at hand at present, then life ceases because its governing, controlling centers are inherently prevented from continuing their functional activity.

These conceptions concerning meningitis have been won because of the operation of cisternal drainage, and the careful study of the conditions which resulted from the operation. Inasmuch as this newly acquired knowledge may serve as a stepping stone toward the eventual solution of these additional problems, the operation of cisternal drainage may yet mark an epoch in the effort to combat the ravages of the disease.

The operation should not be discarded entirely. It may prove a useful measure in selected cases, and if the other factors entering into the problems presented in meningitides come to solution, this means of operating will probably find renewed use because of the easy access it permits to the parts affected.

The author concluded by expressing the optimistic belief that the right road is being followed, that the surgical treatment of meningitis has advanced the comprehension of the disease, and that eventually means will be found to lessen the mortality percentage in this grave condition.

DR. EWING W. DAY, Pittsburg, thought the only thing that had been accomplished so far by the work under discussion was to overthrow some of the existing theories. Cushing's theory, to the effect that death is caused by pressure and that if the pressure is eliminated the disease can be overcome, had been entirely overthrown. The cerebral circulation was not sufficiently well known for one to understand the matter. The work of Leonard Hill, of London, on the circulation was important in this connection. The easiest and most direct tract was through and down the posterior part of the brain. The place where the maximum amount of pus was found in all his postmortems was back of the optic chiasm in the region of the cistern of the chiasm. He had thought there were probably some places where the circulation is very sluggish, and that around the cisterna, back of the chiasm, where it supports the medulla, the circulation probably moves considerably more

slowly than it does in other portions of the brain. It was in such places that pus was found in quantities sufficient to amount to an abscess. It seemed to be demonstrated that the space in front of the medulla could not be drained. That seemed not to be affected by drainage. He believed Dr. Kopetzky right when he said there is sepsis going on in all cases of meningitis. Almost all the cases, following drainage, ran a markedly septic course, with daily variations in the temperature, ranging from normal to 104°. In one case sinus thrombosis was suspected and the sinus was opened, with negative results. In some cases the disease ran a typical septic course, the patient going off into coma, as in advanced typhoid, with muscular relaxation, loss of control of sphincters, dilatation of the pupils, and gradually going off into death. This was not the course in all instances, but in the majority.

Referring to the case cited in Dr. Bryant's paper, in which streptococcus serum was used, the speaker said all the symptoms of meningitis were present, yet the patient recovered. He did not believe the treatment had any effect in producing a cure—he thought nature did it. He did not believe there is any known agent or method which will cure an active meningitis.

DR. FRANCIS P. EMERSON, Boston, emphasized the fact that two principles are to be observed in the surgical relief of any septic affection: First, the establishment of adequate drainage; second, the removal, as far as possible, of the diseased focal areas. The second problem in the surgical treatment of meningitis was impossible. With regard to the first, it was important to establish drainage early in the course of the disease, before the toxemia and sepsis have lowered the vitality of the patient. Theoretically it might be better to relieve the pressure by reversing the lymph current. He thought a study of the autopsy findings would cause one to hesitate about operating upon any case, especially those similar to the admirable group which Dr. Day presented last year before this society. Some cases have recovered without surgical intervention and others have been cured by operative treatment. In the former class of cases the cerebrospinal fluid was sterile. The question of diagnosis with reference to deciding whether or not to operate was important. The symptoms which led to the clinical diagnosis of meningitis did not tell whether

one has to deal with a circumscribed or a diffuse meningitis. Even should an active organism be found in the cerebrospinal fluid, it was necessary to cultivate it before a decision could be reached as to its virulency. Dr. Kopetzky's work, therefore, was important in stimulating renewed interest in the subject, that there may be more exact data for operative interference. His personal feeling, however, was that, so far as relief from surgery was concerned, sufficient advance had not been made in securing such data, and the results from surgery had not been sufficiently satisfactory to warrant the advocacy of any particular line of operative intervention.

Dr. J. S. KIRKENDALL, Ithaca, had been interested recently in the report of a case from a young physician in Ithaca who is well versed in pediatrics. Dr. W. L. Van Pelt told him that a young child had tuberculous meningitis soon after birth, its weight being only nine pounds. He gave this infant twenty-five grains of urotropin daily and the child recovered.

Dr. WENDELL C. PHILLIPS, New York City, was interested in the case mentioned by Dr. Kirkendall, and asked if the cerebrospinal fluid was examined.

In Dr. Bryant's paper, descriptive of a case of purulent meningitis, the essayist had mentioned that the patient had a pulse rate of forty. A pulse rate of forty had never been observed by Dr. Phillips in an uncomplicated case of purulent meningitis.

Dr. JOSEPH C. BECK, Chicago, had always understood that the pneumococcus was the least virulent of the organisms found in meningitis, and had thought his case of meningitis which recovered was that in which this organism was present. He had been working along this line for a number of years. He had had two cases within the last year in which he operated by the Haynes method. Both patients died, but he had learned a good deal from them. In one case of cerebrospinal meningitis he gave urotropin, in doses of one hundred and eighty grains a day, then withdrew ten cubic centimeters of blood and allowed it to settle to form a serum, which he drew off, centrifuged and injected four cubic centimeters intraspinally, as suggested by Swift and Ellis with salvarsan in syphilis. The result was that he had a very irritable patient, with a very clear mind, for three days, but the man went on to death just the same. In another case he used collargol

intravenously. There was a distinct change in the fundus of the eye in the form of a pigmentation.

DR. DAY, continuing the discussion, said he was asked by one of the surgeons why he did not drain the space in front of the medulla. He did that in one case, placing the drain underneath the dura, passing it up to the cisterna, back of the chiasm, in front of the medulla. The patient went on to death just the same. He had to go inside the dura, and the cerebellum was crowded up, cutting off the drainage. The only other possibility was to put in a lead tube.

DR. THOMAS J. HARRIS, New York City, said Dr. Bryant had closed his paper by expressing hope for the class of cases under discussion, and yet had cited only one case in which there was recovery. From a careful study of the literature it was the speaker's opinion that many such cases have been reported. One should be encouraged by well authenticated cases which recover. One or two cases had been reported in New York which, he felt perfectly sure, were cured. The Germans made their diagnosis upon the findings in the cerebrospinal fluid, pronouncing the case one of meningitis when the meningococcus was found in the fluid. One German clinic had reported forty cases of cured recurrent meningitis. This should be encouraging to all who are working along these lines.

DR. SAMUEL IGLAUER, Cincinnati, called attention to the importance of some experimental work on the circulation of the cerebrospinal fluid which Dr. Kramer of Cincinnati had been doing. This might readily explain the failure of the Haynes operation to provide adequate drainage in otitic meningitis. According to Kramer, circulation of the cerebrospinal fluid below the tentorium is separate and distinct from that above the tentorium. If this be true, then drainage through the cisterna magna will fail to relieve the areas commonly involved in otitic meningitis. Kramer has also shown that methylene blue injected in the lumbar region of animals will enter a small pore in the lower portion of the spinal cord and ascend (ciliated epithelium) through the central canal to the fourth ventricle. Through a similar action he explained the fatal effects occasionally noted when serum (with toxic tricrosol preservation) was injected in the lumbar region of children for the relief of epidemic meningitis.

DR. SEYMOUR OPPENHEIMER, New York City, spoke of a case which Dr. Haynes quoted in his original paper. All the evidences of acute meningitis were present, and there were signs of some intracranial involvement. The child was operated upon promptly, the findings being sinus thrombosis, mastoiditis, extradural abscess, and a large area of encephalitis. The patient recovered. Four weeks later meningitis developed. Lumbar puncture was made and streptococcus mucosus found. Six months later the child again went into coma, and again the streptococcus mucosus was found. It had had previously a very short and acute attack of nasal infection. The postnasal infection could easily go through the roof of the aural cavity, the bony wall having been removed at the original operation. Fortunately he obtained a postmortem. He found evidences of an acute meningitis over the region of the middle cranial fossa. This suggested the point that, in view of the fact of it being impossible to differentiate clinically between circumscribed and diffuse meningitis, it is probable many cases reported as cured are cases of very circumscribed suppurative meningitis.

The Observation of Nystagmus Through the Closed Eyelids.

DR. EDMUND PRINCE FOWLER, New York: The anterior portion of the eyeball is the segment of a smaller sphere than the posterior portion, and consequently the cornea projects from the sclerotic somewhat in the same manner that a water glass does from its case. In different people and at different ages the degree of curvature varies, but at all times it is sufficient to enable one to follow movements of the eyeball in every direction, though the eyelids be closed. By observing nystagmus through the closed eyelids the following advantages are gained: (1) The eye muscles are at rest, and no accommodation or fixation occurs. Thus all patients may be tested under equal conditions in so far as these uncertain and disturbing elements are concerned. (2) There being no effort at fixation, after rotation, nystagmus endures from twice to three times as long as when the eyes are opened following rotation. Thus a longer, and hence a more accurate, guide is furnished for the rotation tests, and nystagmus may be aroused by weaker galvanic and caloric stimuli. (3) During vertigo there is an involuntary tendency to close the eyes and to keep

them closed, as this lessens in some measure the dizziness. (4) By the use of suitable indicators, properly attached to the eyelids, the eye movements may be magnified a hundred-fold, thus making for ease and accuracy of observation. (5) Upon properly placed revolving drums, or moving sheets, such indicators may be made to graphically record nystagmic movements of both eyes, during and after rotation of the body.

A means is thus provided wherewith to estimate not only the duration of nystagmus during and after rotation, and the caloric and galvanic tests, but to study the magnitude and frequency, gradations and variations of nystagmus in both eyes under different conditions. Such a study cannot fail to contribute something to the understanding of vertigo and allied phenomena.

A study of the modifications of nystagmus in disease is made possible by the means suggested, and a nystagmogram may be preserved for future reference.

In order that inertia and momentum may be practically nil, the author has used paper or straw indicators, one end stuck to each eyelid, usually to the nasal side over the junction of the cornea with the sclera. Zinc oxid plaster or auto tire cement are satisfactory adhesives.

One disturbing factor is the winking or twitching of the eyelids. This is clearly recorded upon the tracings. After a little practice these may be easily distinguished from the nystagmus oscillations.

For making the nystagmographic tracings a sphygmograph mounted upon a hocky puck is used, the latter being held between the teeth of the patient, and the blackened paper ribbon fed from the rear towards the front, away from the patient.

DISCUSSION.

DR. ARTHUR B. DUEL, New York City, thought Dr. Fowler had presented a very ingenious method of making permanent records of ocular phenomena in vestibular nystagmus. He did not think such observations necessary to diagnosis, but when the apparatus was perfected the records, for those who understood them, would be useful in reporting cases. The tracings of nystagmus would leave no doubt of its presence. When one must depend upon the house staff to report cases

that arrived during one's absence, these tracings could be made and put into the records for subsequent study.

DR. FOWLER, in closing the discussion, said patients did not notice as much vertigo with rotation or following caloric reactions with the eyelids closed as they did with them open. For instance, one could produce nystagmic movements of the eyes with caloric stimulations and stop before the vertigo came on. Irrigation usually makes patients deathly ill, and it surely is a great advantage to be able to avoid this annoying feature of the labyrinth tests. In many neurasthenics the rotation tests show nothing on account of the incessant blinking and rolling of the eyes around and about. If the lids are closed some approximation to the duration of the nystagmus may be ascertained.

SYMPOSIUM: AURAL COMPLICATIONS OF THE EXANTHEMATA.

(a) Etiology, Diagnosis and Treatment.

DR. CHARLES R. C. BORDEN, Boston: The present communication is practically a continuation of a paper presented at the Ninth International Otological Congress, in Boston, in 1912, based upon a study of the aural conditions found in four hundred and fifty-four autopsies after death from diphtheria, scarlet fever and measles. Only aural complications of exanthemata are considered, and not aural diseases in general, and only severe cases, such as are seen in hospital practice, not the mild variety usually encountered in private practice.

Aural complications in the exanthemata occur in from five to forty-four per cent of clinical cases, as stated by different writers. In fatal cases this percentage was found to be greatly increased. In the autopsy reports upon which the paper is based, the percentage in diphtheria was eighty-two, in scarlet fever ninety-four, and in measles one hundred per cent.

One would naturally expect unrecognized and untreated middle ear inflammation or mastoiditis to go on to meningitis, brain abscess, and jugular thrombosis, but the autopsy records showed only four cases of septic meningitis (not necessarily of middle ear origin), one case of infection of the jugular vein, and not a single case of brain abscess. Complications in

the heart, pleura, joints, and kidneys, on the contrary, occurred with startling frequency. While it could not be held that all of these complications arise from middle ear disease, a certain number are. Age is usually an important factor. Generally speaking, the younger the patient the greater the susceptibility to aural complications.

The symptomatology of the aural complications in the different exanthematous diseases is given in brief detail. The most striking variation between the three diseases, diphtheria, scarlet fever and measles, is in the appearance and characteristics of the pus found in mastoiditis at autopsies in diphtheria. In scarlet fever and measles it is of the ordinary variety and is usually copious in amount. In diphtheria it is repeatedly described as green, yellowish green, brownish green, or other colors bordering upon a greenish hue, and as being thick, tenacious, gummy, gelatinous or semisolid, etc. The marked peculiarity of the discharge may largely account for the lack of active symptoms which seem to be peculiar to aural complications of diphtheria.

As the infection, in aural complications of contagious diseases, reaches the middle ear by way of the eustachian tube, and the nose and nasopharynx are the seat of active inflammations, these would seem to be the structures to which preventive measures would be aimed, but experience and practice have proved otherwise. Irrigation of the nose and nasopharynx, which was formerly so commonly practiced, is now considered, by the physicians and aurists at the Boston City Hospital, to be absolutely contraindicated, under any circumstances, and this includes all sprays and douches, as well as aural irrigations.

There is but one true method of preventing aural complications, viz., through the removal of adenoid tissue before the patient contracts a contagious disease.

Inasmuch as aural complications are usually well established before attention is called to them, the treatment is almost entirely surgical. In general it is the same as in ordinary aural practice, with one or two exceptions. The important difference is the low vitality of the patient and the increased virulence of the infection. Nature is to be relied upon less in such cases than at other times. Middle ear inflammation which might be aborted under other circumstances

fails to respond to the usual mode of treatment in contagious diseases. The ordinary methods of treatment are not available. If the drum membrane is found to be red and inflamed, paracentesis is indicated. Hot irrigations seldom accomplish the desired results. Inflation of the middle ear, and postnasal applications, not only avail nothing, but decidedly increase the inflammatory process. The inhalation of hot steam is a safe procedure, but usually fails to give relief. Free paracentesis is the only accepted method of procedure, and should not be delayed. Hot irrigations are advantageous. As the discharge becomes less in amount it is often better to discontinue the irrigations and to substitute warm peroxid.

The sudden cessation of a discharge that has been profuse should at once direct attention to the mastoid. Whenever the middle ear is suspected of being the primary source of infection in complications of a serious nature and free paracentesis has failed to control the aural inflammation, the mastoid should be opened at once.

Summing up the treatment of aural complications in exanthemata, two methods are efficacious, viz., hot irrigations and paracentesis.

(b) The Etiology, Diagnosis and Treatment of the Aural Complications of the Exanthemata.

DR. STANTON A. FRIEDBERG, Chicago (by invitation): In considering the etiology of the aural complications arising during the course of the exanthemata, it may be broadly stated that their number and severity will depend upon the class of patients and the character of the epidemic. Other factors that must be taken in the light of predisposing and contributing causes are age, climate, season, physical condition of patient, condition of the upper respiratory tract, the specific infective disease, cross infections and systemic complications.

There can be no hard and fast rules established with reference to the diagnosis of the aural complications of exanthemata. In most cases conclusions in regard to the aural condition are reached in the same manner as in the ear infections from other causes.

The treatment of aural complications naturally divides itself into preventive and curative. The preventive treat-

ment aims at the alleviation of the accompanying nasal and pharyngeal conditions. Early treatment depends upon the conditions present. Paracentesis and hot irrigations are advocated. The maintenance of drainage is important. In cases in which the aural discharge persists for several weeks, despite the most careful attention, consideration may be given (1) to the use of vaccines; (2) to the correction of conditions in the upper respiratory tract which contribute to a continuance of the infection; (3) to the mastoid operation to provide for better drainage from the antrum and middle ear cavity.

The treatment for the more serious complications does not differ from that pursued in cases arising independent of infectious diseases.

In concluding, the author emphasizes the following points: (1) The closer cooperation between the attending physician and the aurist in private practice. (2) The necessity of competent aural surgeons being in attendance in contagious disease hospitals. (3) The isolation of patients in these hospitals at least up to the period of convalescence, lessening the liability to cross infections. (4) The necessity of the closest attention to patients, which includes routine examination of the ears in at least the younger patients, and careful observation and treatment when symptoms of aural extension arise.

DISCUSSION.

DR. HENRY O. REIK, Baltimore, considered the papers of Dr. Borden and Dr. Friedberg as two of the most important presented before the society for some time. He congratulated the authors upon the very complete manner in which they covered the subject, the complete way in which they had digested the various facts, and the conclusions which they had brought out. He thought the matter might well be brought more closely to the attention of the general physician, and he hoped each author would take it upon himself to force it home to those who have charge of infectious disease hospitals. Dr. Friedberg had called attention to the importance of this in referring to the frequency with which aural infection takes place in infectious diseases. Dr. Reik had recently observed, in one large hospital in which four hundred beds were allotted to diphtheria and scarlet fever, that practically no attention was paid to the middle ear. Tympanotomy was

rarely performed, and the ears rarely examined; the attendant waiting until the child complained of pain. Practically one-third of all the cases had spontaneous perforation and otorrhea, and a large percentage of these went out of the hospital with discharging ears. The number of complications which resulted could not be estimated in figures. In a similar hospital in Baltimore the same conditions pertained in 1912. With the advent of a new superintendent in 1913 the situation was changed. An otoscopic examination was made in every case admitted, and three examinations daily after admission, one in the morning, one at four o'clock, and one at night. Thirty-seven patients had developed some indication of otitis media. The examiner was very keen to observe this condition, and if there were any doubt as to the need of paracentesis he would operate. Each one of the thirty-seven had tympanotomy, and each child was dismissed with dry ears. In one hundred and ninety-one cases of scarlet fever every patient went out without otorrhea. That showed what could be done with careful observation and examination.

Dr. Borden referred to mastoiditis without tenderness. The speaker called attention, in this connection, to the fact that there is an instrument for measuring the presence and amount of tenderness, or rather the amount of pressure necessary to elicit tenderness, which is much more accurate than measuring it with the thumb and finger. This algesiometer was a very simple device, consisting of a rod working in a cylinder, with a spring arrangement for measuring grams of pressure. This apparatus could be placed directly over the antrum or over the tip cells. With this instrument it was often possible to elicit tenderness which the thumb and finger would not detect.

DR. JOSEPH C. BECK, Chicago, said he staid away from these cases as much as possible. In Cook County Hospital, in Chicago, there was great neglect of patients of this class because men who were able to take proper care of them did not like to treat them. This was because of the danger of carrying the infection. He has had this experience in his own family, infecting his own child after treating a hospital patient. He could say definitely that there was no other exposure in this case. Every large hospital should have a contagious ward, in charge of physicians and nurses specifically trained for these diseases. Attending physicians should be called only

when absolutely necessary. Another point to which he wished to refer was the matter of operating upon the nasopharynx and tonsils while the patient is still in the hospital. He had seen a large group of cases operated upon with excellent results, particularly with reference to recovery from the nephritis which is going on without typical findings in the urine or other manifestations elsewhere in the body. In a large hospital where operations could be performed under the most advantageous circumstances, especially with reference to the anesthetic, very satisfactory results could be obtained. He thought the open method of anesthesia a distinct advance. With reference to postmortem changes in the ear, Bezold's latest statistics showed that these changes are postmortem unless the autopsy is very promptly made.

DR. J. S. KIRKENDALL, Ithaca, asked Dr. Borden how he dared to give one of these patients ether after kidney complications.

DR. WILLIAM B. CHAMBERLIN, Cleveland, considered the papers presented by Dr. Borden and Dr. Friedberg very important. He mentioned a case of aural complications of scarlet fever which had come under his observation during the winter. The patient was a man, thirty-five years of age, whose drum membrane had ruptured spontaneously without premonitory symptoms. He was still in bed, the scarlet fever infection having almost spent itself. There was still a profuse discharge from the ear, but no signs of mastoiditis. One morning the nurse noticed a swelling behind the ear. He operated; finding the most extensive mastoid process he had ever seen and every cell filled with pus and granulation tissue. He called attention to the value of the X-ray in making a diagnosis in the class of cases under discussion. Dr. Ingersoll and he, at the Lakeside Hospital, had X-ray examinations made in all cases. In chronic cases he did not rely so much upon it, but in acute cases it was of the greatest value. In questionable cases he thought the simple mastoid operation would do no harm if there proved to be no mastoid involvement, and was attended with little danger. Should there be mastoid involvement, failure to operate would do great harm.

DR. A. P. VOISLAWSKY, New York City, asked if the essayists were able to stop the aural discharge. He had had a

great deal of trouble in having to keep children week after week on account of his inability to check the otorrhea.

DR. GEORGE M. COATES, Philadelphia, referred to the value of vaccine therapy in the management of the conditions under discussion. He called attention to a report of McKernon, made to the society in 1910, in which he stated that by the use of autogenous vaccines in cases of mastoiditis following measles and scarlet fever upon which he had operated, the time for wound healing, which is usually much prolonged in these cases, was reduced to the normal. A report of Weston and Kolmer, in 1911, shows the results of one hundred cases of suppurative otitis media (scarlatina) treated with autogenous vaccines. Their work was done in the Philadelphia Hospital for Contagious Diseases. Under old methods of treatment, according to the histories of many hundreds of cases, it was found that the percentage of dry ears obtained under thirty days was only 7.46. With the use of bacterins this was increased to 22.9, which is a considerable gain and a fair index of the value of bacterins in this class of cases. Undoubtedly more dependence will be placed on this method in the future.

DR. DUNBAR ROY, Atlanta, agreed with Dr. Borden with reference to the irrigation of the nasal chambers in exanthematous conditions, believing that it produced more irritation and gave rise to more possibility of infection of the middle ear than if left alone. In the later stages, when the discharge was very thick and mucopurulent secretion came from the lateral sinuses, it was sometimes pitiful to see young children trying to get air through the nasal passages. So long as the secretion existed it rendered them more liable to infection of the middle ear. In such cases he used a small rubber tube with a bulb at one end. By inserting the free end of the tube into the nasal passage of one side and blowing air through it he had found it possible to blow through the opposite side large quantities of secretion. The secretion was thus prevented from going up into the eustachian tube. The child was rendered much more comfortable by clearing out the nasal chambers in this way.

DR. H. HOLBROOK CURTIS, New York City, thought it would be better to suck the secretion out than to blow it up in the manner described by Dr. Roy.

DR. TALBOT R. CHAMBERS, Jersey City, spoke favorably of the Yankauer nasal speculum, through which he had relieved the discharge in a number of cases of eustachian catarrh, by means of iodine applications.

DR. NORVAL H. PIERCE, Chicago, emphasized the importance of early paracentesis in these cases. It was absolutely necessary to resort to paracentesis early in order to prevent mastoid bone complications. If one remembered the anatomy of the regions involved—the aditus, the cavum, the pneumatic spaces, all communicating by minute tubes with the antrum—it would be easy to see that paracentesis can prevent involvement of the mastoid bone only when done early. At the very inception of otitis media the infection spreads immediately down these tubes, and unless the paracentesis be done at a point in the course of the disease before the mucosa of the tubes swells, one would be unable to draw off the infectious material by capillary drain or otherwise, and it would dam up in the pneumatic spaces. The mucoperiosteum, as was well known, has the power of swelling enormously, no other tissue in the body having this power to an equal extent, nor of exercising it so suddenly. In twenty-four hours it could swell sufficiently to fill a large pneumatic space. Paracentesis, therefore, did most good when performed early. It was the retention of discharge and the swelling of the mucoperiosteum that produced decalcification of the bone and consequent mastoid involvement.

DR. THOMAS J. HARRIS, New York City, thought this symposium one of the most important which had come before the society in many years. He regretted that the general practitioner could not have joined in the discussion. He asked the essayists, in closing the discussion, to speak particularly of the prevention of the complete destruction of hearing following the exanthemata. Milligan, of Manchester, had advocated the mastoid operation of postauricular drainage in these cases.

DR. FRANCIS P. EMERSON, Boston, advocated frequent inspection and, if in doubt, incision of the drum. He cited a case in which the patient, when first seen, had no other symptom than fever. He decided to wait, and four hours later the drum ruptured spontaneously.

DR. FRIEDBERG, in closing the discussion, maintained that

the majority of acute otitis cases would recover if properly handled. The result depended largely, as Dr. Borden had said, upon the virulence of the infection and the vitality of the patient, but ordinarily the percentage of recoveries was large. In the series reported there were two cases out of thirty-five dismissed from the hospital with discharging ears. This showed what could be done by proper attention.

The Exploratory Opening of the Sphenoid Sinus.

DR. CHARLES PREVOST GRAYSON, Philadelphia: By this exploratory opening is meant one that can be made so extemporaneously, with so little discomfort to the patient, so little derangement of his ordinary pursuits, that it may be employed for merely exploratory or diagnostic purposes. The artificial opening advocated is made on the anterior wall of the sphenoid sinus, at a point as close as possible to the angle of junction of its floor with its internal wall. As regards the safety and facility with which it is made, this opening is on a par with the puncture of the nasal wall of the antrum beneath the inferior turbinate, or with the simple enlargement of the ostium frontale by means of the rasp or other instrument. This opening can be utilized for both exploratory and therapeutic purposes, and it has the advantage of not involving either the destruction or the crippling of any of the functionally valuable intranasal structures.

The technic of the operation is as follows: The inner or nasal portion of the anterior surface of the sphenoid body is exposed as widely as possible by shrinking the turbinates with one of the adrenal preparations. The field of operation is anesthetized with cocain and then rendered ischemic by the adrenal solution. When this has been done the course of the sphenopalatine artery is usually so distinctly visible that it can be readily avoided. The application of a dilute tincture of iodine will be sufficient for purposes of sterilization. The instrument with which the sinus wall is perforated is a straight drill, tipped with a conical bur six millimeters in length and measuring two and one-half millimeters from its point to its greatest diameter. The drill is applied two or three millimeters above the line which divides the anterior from the inferior surface of the sphenoid body and close to the attachment of the ethmoid plate in the middle line. The opening

it makes is two millimeters in diameter, which is quite sufficient to permit the escape of any fluid within the sinus, the introduction of an appropriate irrigation cannula, or, should it seem advisable, the distal jaw of a biting forceps with which the opening may be enlarged. If the exploration of the sinus proves to be pathologically negative, the breach will close within twenty-four hours.

In closing, the author reiterated that his object in exploiting this method of investigating the sphenoid sinus is, in the first place, to dislodge the idea that the ostium sphenoidale should always be the starting point for any operation upon the sinus; to lessen, if successful in this, the frequency with which the middle turbinate is unnecessarily removed, or, in other words, to substitute for a somewhat formidable and tissue destroying operation one that is technically simple and unattended by any loss of functionally useful tissue; to lessen, also, the hesitation with which some thoroughly qualified men contemplate the surgical invasion of this sinus.

DISCUSSION.

DR. H. HOLBROOK CURTIS, New York City, defended the usual operation of opening the sinus through the natural orifice, for the reason that the frequent excursions of the cell described by Sieur and Jacob, impinging on both the sphenoid and antral walls, might, by injury, lead to an infection, and because the cell overhung the sphenopalatine fissure and ganglion, these structures, as well as the optic and superior maxillary nerves, might be injured in case Sieur's cell was entered by accident and infected. Dr. Curtis then went into the question of opening the inferior face of the sinus, which Dr. Grayson explained he had not advocated, and the remarks were withdrawn.

DR. ROSS HALL SKILLERN, Philadelphia, fancied, he could hear Dr. Grayson saying to himself, "How long, O Cataline, wilt thou abuse our patience?" He was not in accord with the essayist as to the indications for, or the method of opening the sphenoid. Unless purulent secretion were present in the sphenothmoidal fissure in frank cases, or the typical pressure symptoms were present in the closed and latent type, he did not perceive the indication for exploratory opening. As to the method, he preferred the one which is constantly under control of the eye. This was nearly always possible

in the presence of disease, for it was a well known fact that a diseased sinus is always easier to sound than a healthy one, this being due to the enlargement of the drainage passages by the constantly outflowing secretion. In his experience this was peculiarly adaptable to the sphenoid. Under these circumstances, after the sound had found the ostium and has penetrated into the sinus, it was a simple matter to introduce a small Hajek curette or an evulsor and to make a comparatively large opening in the anterior wall and at its thinnest part. All danger of penetrating the cribriform plate or completely missing the sinus was obviated. It would seem that this is really the safest and sanest method of approaching this cavity for diagnostic and therapeutic purposes.

DR. FRANK R. SPENCER, Boulder, said one could easily use the Andrews probe to find the opening. It was perfectly justifiable to remove the posterior half or one-third of the middle turbinate in order to expose the sphenoid cavity. The thin anterior wall could be broken down and an opening gained which would be large enough for therapeutic purposes. That could be done without cocaine anesthesia in simple cases.

DR. JOHN O. ROE, Rochester, said: "It is not difficult to find the natural opening of the sphenoidal sinus, although I think Dr. Skillern had located it somewhat lower than I have generally found it. My own method of locating the opening is to pass the probe along the lower border of the middle turbinate, using it as a guide, then by turning the end of the probe slightly upward, the sphenoidal opening is readily entered. In some cases the opening can be seen by anterior rhinoscopy when the middle turbinate is small. When the opening has been found, the cavity can be explored in every direction and any abnormalities dealt with as conditions indicate. Since suppurative conditions are those most commonly found in these cases, free drainage of the cavity, as pointed out by Dr. Grayson in his excellent paper, is of the utmost importance. This I have established most easily by taking away the lower wall with forceps, cutting downward in an anteroposterior direction. In the removal of this bone, however, I have not often found it so slender and fragile as Dr. Grayson has indicated, but, on the contrary, usually quite hard and dense, sometimes requiring the use of the chisel.

"I might relate, in this connection, that a few years ago there came under my observation an exceedingly interesting

case of tic douloureux, due to a myxomatous growth, occupying the entire cavity of the sinus, and on the removal of this growth the tic douloureux subsided.

"When we consider the great anatomic variations in different skulls, in no case would I attempt to drill or chisel an opening into the sphenoid sinus without first having found the natural opening to serve as a guide for the operation."

DR. TALBOT R. CHAMBERS, Jersey City, thought it better to start with the natural opening, enlarging it as much as necessary, rather than to make a second opening.

DR. GRAYSON, in closing the discussion, could only repeat that in his opinion the chief objection to the usual method of opening the sphenoid sinus was that it was begun in what was concededly a region of risk instead of being cautiously ended there. He thought it better from every possible point of view to begin the operation at the point he had designated. Anyone familiar with the normal anatomy of the sinus, as well as with its occasional abnormalities, must admit that this is the safest locality not only at which to enter it but from which to begin the removal of its anterior wall. The terms thin and thick which had been applied to this wall were purely relative, and when one spoke of its lower being thicker than its upper portion it meant no more than a difference of one or two millimeters, which was certainly of no surgical consequence whatever. It was scarcely conceivable that anyone with the delicacy of touch that the rhinologist should possess could inflict any injury through this operation. Its greatest merit, in fact, lay in its freedom from any unnecessary or concomitant injury. The opening was made under the direct inspection of the eye, and there was no flow of blood to obscure the field of operation. In the large majority of cases it was necessary to remove no more than the inner portion of the anterior wall, and he had yet to hear a single good reason for continuing the ablation of the middle turbinate in order that we might begin our sphenoid operation at its awkwardly and, comparatively, dangerously placed ostium.

A New Technic for the Removal of Intrinsic Growths of the Larynx.

DR. ROBERT CLYDE LYNCH, New Orleans: As perfect quiet is of the greatest necessity, the author insists on his patients being kept continuously in the surgical stage of anesthesia,

securing perfect relaxation of the parts, conducing to the most accurate work. Having obtained a perfect view of the larynx (using his modification of the Killian and Albrecht suspension apparatus), with that organ and its owner quiet, he proceeds as follows: In vocal nodules the affected cord is picked up gently, turned to nearly an angle of forty-five degrees, in order that the under surface may be seen. The Killian baby forceps and Killian baby double cup forceps are used according to the size of the nodules. If the growth occupies the superior surface and is seen to involve mainly the subepithelial structures, the surface layer is split with the knife and the small tumor picked out with appropriate forceps. The surface membrane is reapplied and the wound dressed with tincture of benzoin compound and collodion. In single pedunculated tumors the tumor is picked up with the forceps, encircling the base with a wedge shaped incision, removing it by clear dissection with a knife. Single papillomata are grasped with forceps and shaved off below the level from which they spring. In the case of multiple papillomata the mass is grasped with Killian baby forceps and the entire area clearly dissected. Lest some small portion might be left behind, the whole surface is gently curetted, including the subglottic area.

Intrinsic epithelioma of the larynx is removed completely, by dissection, through the mouth, the tumor being delivered in one mass upon a cartilaginous plate. A case cited by the author was the first on record, so far as he knew. The diagnosis and the fact of complete removal were verified by the microscope. By this method the tumor is removed in one mass without itself being disturbed by instrumentation or manipulation, which is the accepted surgery of malignant growths, thus diminishing the danger of recurrence.

The Proper Fields of Medicine and Surgery In Diseases of the Upper Air Passages.

DR. JOHN A. THOMPSON, Cincinnati: One-half of all the diseases of the upper air passages are curable by medicinal means alone. An intelligent use of known methods in medicine will often prevent complications that make operation necessary. The common diseases of the nose and throat are easily separated for treatment into three classes: First, those the

treatment of which is purely surgical, such as deformities, deflections of the septum nasi, chronic sinusitis, all tumors, benign or malignant, adenoids, hypertrophied tonsils, quinsy, retropharyngeal abscess, foreign bodies, hemorrhages, and stenosis of the larynx. In some of these, preliminary treatment makes the operation easier and the recovery surer.

The second class includes diseases where combined medical and surgical treatment is necessary. The most important, because the most frequent, disease of this class is hypertrophic rhinitis. After removal of the newly formed connective tissue in the turbinates, an analgesic germicide that will at the same time relieve congestion is indicated. Menthol meets these requirements. Camphor is similar to menthol in its local action, and can be advantageously combined with it.

Other conditions which require combined treatment are tertiary syphilitic ulcer, chronic granular pharyngitis, and edema of the glottis.

The third class is chiefly represented by acute rhinitis. Other conditions which may be treated by medicinal measures alone are acute catarrhal sinusitis that often accompanies acute rhinitis, acute laryngitis, simple chronic rhinitis, chronic purulent rhinitis, atrophic rhinitis, chronic laryngitis, chronic tracheitis, and chronic bronchitis.

The treatment ordered to abort an acute attack, with variations to suit the individual, is first a sweat, however procured. To open the nose blocked by the swelling, a solution of adrenalin may be used every three hours. A saturated solution of boric acid may be advantageously employed to dilute the adrenalin. Where the patient is seen in the second stage of the disease and the serous discharge from the nose is annoying, a spray containing one grain of atropin to two ounces of liquid petrolatum is very effective.

Two cases of Vincent's angina successfully treated with salvarsan in glycerin were cited.

Emphasis was laid upon the utilization of simple means, by which much can often be accomplished in various affections of the upper air passages.

DISCUSSION.

DR. JOSEPH H. ABRAHAM, New York City, regretted that he had come in too late to hear all of Dr. Thompson's paper,

but he heard what was said with reference to Vincent's angina, in which connection he wished to present a remedy which he had used in four cases of verified Vincent's angina. It consisted of pure carbolic acid, fused, and applied, upon a cotton tipped applicator, to the ulcerating surfaces. Two applications were made a day, one when the patient came in in the morning and the other in the afternoon. The acid was allowed to remain in contact with the tissues from two to five minutes, and then neutralized with pure alcohol. The patient was given a simple cleansing mouth wash to use at home. In three cases, when the patient was sent to the pathologist the next morning, no bacilli and no ulceration could be found. In the fourth case a few scattered organisms could be found in the tonsils and quite a number in an ulcerated tooth socket. He removed the root of a tooth, curetted the cavity, and applied carbolic acid, and the next morning there was no culture. In each case the acid was used twice. Subsequent examination failed to reveal any bacteria, and the patient was dismissed with a cleansing wash.

DR. LEE M. HURD, New York City, considered intratracheal injections of various medications with oil one of the best methods of treating these inflammations. Intratracheal injections were not employed as much as they should be. In Vincent's angina any acid would do—trichloroacetic, strong nitric, or any acid or strong caustic. The spirillum would not be found the next day. In very severe cases salvarsan, as suggested, was good. The oily injections would relieve the chronic laryngitis, and was useful in chronic and acute inflammation of the trachea. Sweet oil or petrolatum, about two drams, with some medication, injected into the trachea and bronchi had been found efficacious.

DR. TALBOT R. CHAMBERS, Jersey City, referred to the theory of Sir W. Arbuthnot Lane, of London, concerning the use of petrolatum. According to this theory, petrolatum passes through the intestine and is not absorbed. Dr. Hurd advocates, in consonance with Dr. Thompson, the injection of two drams into the bronchi; Dr. Chambers would like to know what becomes of that oil. If not absorbed, it would become a foreign body.

DR. HURD, replying to Dr. Chambers, said the vaselin which he used was probably absorbed.

DR. GEORGE L. RICHARDS, Fall River, thought the demonstration of Dr. Beck of very much greater value to laryngologists than theorizing about the absorption of this or that oil. He had been especially impressed with the picture showing the blood vessels, which illustrated so clearly why hemorrhage takes place after these operations. The sections demonstrated also why there is infection and more or less mucopurulent periostitis after these operations. Work such as Dr. Beck had presented led to more exact knowledge. Therapeutics was largely a matter of theory, but this histopathologic work was certainly conducive to exact knowledge.

DR. GEORGE F. COTT, Buffalo, recalled that in 1901 Dr. Thompson had read a paper on this subject. Since that time he had followed the suggestions then given, with very good results.

DR. THEODORE CORWIN, Newark, thought the intratracheal injections most valuable. Patients could be taught to make the injections themselves, using a long dropper and injecting fifteen or twenty minims two or three times within ten minutes and repeating this every half hour or hour, so long as the cough was annoying. He used vaselin or other oil in combination with menthol, one or two per cent, camphor one per cent, or anything that might be desired. For office treatment the tracheal syringe was preferable, giving doses of one or two drams. It should be preceded by a downward spray of two per cent menthol to render the larynx less sensitive to manipulation with the syringe.

DR. RICHARDS added that some years ago he used oils of one kind or another, and instructed his patients to use nebulizers. He had reached the conclusion that oils are nearly valueless. It was better to employ something corresponding as nearly as possible in specific gravity to that of the normal serum.

DR. THOMPSON, in closing the discussion, said that when Dr. Harris sent out his circular letter asking for suggestions concerning this meeting, he thought it wise to have several papers concerning the treatment of the diseases of the upper air passages. Dr. Beck agreed to discuss the scientific side, while he took the therapeutic side of the question. He had endeavored to discuss the matter from the point of view of everyday practice. The most important point, and one which

he would reiterate, was expressed in the opening sentence of his paper: "One-half of all the diseases it is our daily work to treat, are curable by medicinal means alone."

DR. BECK, in closing the discussion, said about five per cent of his cases were amenable to nonsurgical treatment. He had reference to diseases of the upper respiratory tract, not to the trachea.

Tuberculosis of the Middle Ear.

DR. H. H. BRIGGS, Asheville: The frequency of tuberculosis of the middle ear in persons suffering from tuberculosis elsewhere in the body has been placed at twenty-five per cent. Of fifteen hundred school children examined by Westmacotte, two per cent were found to have tuberculosis of the middle ear. The disease is probably of far greater frequency than statistics show, and the true diagnosis is often mistaken because its onset is so insidious that attention is not easily called to the condition and no observation is made. Moreover, when the case presents itself the condition usually has passed from that of a pure tuberculous process and becomes a mixed infection, the symptoms of the suppurative condition masking the true nature of the initial disease. The careless manner of classifying all discharging ears as suppurative otitis media, without recourse to the microscope or inoculation tests, is unfortunate.

The middle ear must be regarded as belonging anatomically and bacteriologically to the upper respiratory tract, as insisted upon by Goldstein, who considers primary tuberculous infection of the middle ear of respiratory origin.

Among the predisposing factors may be classed general debilitating diseases, the hereditary influence of tuberculosis, syphilis, association with tuberculous individuals, unhygienic environment, overcrowding, poor food, cachexia—in short, any condition of surroundings or constitution which induces a lowering of the systemic power to combat infection. Among the predisposing causes of more immediate influence may be regarded (1) the existence of a tuberculous lesion elsewhere in the body, especially pulmonary tuberculosis with cavitation, and tuberculous disease of the glandular system; (2) abnormal conditions of the upper respiratory tract, including the presence of nasopharyngeal adenoid growths which have been shown by microscopic examination and inoculation tests to

be the frequent seat of a latent tuberculosis; (3) infancy and childhood offer a predisposition for various reasons.

The channels of infection, are: (1) Mechanical, through the eustachian tube, either air borne or introduced into the tympanic cavity by the aid of particles of mucus or foreign matter during the acts of swallowing, coughing, sneezing, or blowing the nose. (2) Infection along the eustachian tube by other than mechanical means. (3) Through the blood channels. (4) Through the lymphatics. (5) Via the external auditory canal. (6) By extension of an intracranial infection through the internal auditory canal, fallopian canal or the labyrinth. This is mentioned as merely a possibility.

To the author the mechanical theory of infection, especially secondary, seems simplest, easiest and most probable in the great majority of cases. The greatest number of cases occur in early childhood and advanced phthisis, when the conditions favorable to the mechanical passage of infectious material through the eustachian tube are at their maximum.

Clinically two rather distinct forms of tuberculosis of the middle ear manifest themselves—acute and chronic. In each may be found all the changes, from slight infiltration of the mucous membrane to extensive necrosis of the temporal bone. Rapid loss of tissue is characteristic of the acute form, resulting from ulceration of the tubercles throughout the mucosa. In the chronic form the process runs an asthenic course, and infiltration, caseation and necrosis follow less rapidly and with more characteristic tuberculous sequence.

The essential symptom which differentiates tuberculous otitis from other forms is the absence of pain. Even though the destructive process is rapid and the appearance of the membrana per se simulates an acute purulent otitis there is seldom any complaint of pain.

In determining the diagnosis, the family history should be carefully considered with regard to tuberculosis, and the patient's habits, residence, and environment should be ascertained to determine whether there has been an undue exposure to tuberculous persons or unhygienic surroundings. Facial paralysis, especially in children, occurs in one-third of the cases, against one to two per cent in nontuberculous conditions, and is of special diagnostic significance. The sanious and foul condition of the discharge, especially when particles

of bone are incorporated, excites suspicion. Marked impairment of hearing, absence of headache, occurrence of hemorrhage, are considered by some as diagnostic points. Tuberculin injections and blood pressure changes (hypotension) are also considered. The only positive means of diagnosis, however, are: (1) Finding microscopically in the discharge or granulations the tubercle bacillus, or (2) giant and epithelioid cells and caseation in the tissue; (3) by experimental inoculation, reproducing tuberculosis.

The prognosis is, as a rule, unfavorable.

The treatment naturally divides itself into hygienic, dietetic, medical, and surgical, as the case indicates. The use of tuberculin has proved so successful in so many forms of tuberculosis that no tuberculous process in any way localized can be considered invariably to contraindicate its use. The author could see no reason why it should not be indicated and of decided value in properly selected cases of tuberculosis of the middle ear.

DISCUSSION.

DR. FRANK R. SPENCER, Boulder, mentioned a method of making the diagnosis of tuberculosis of the middle ear, consisting of cleansing the canal first, introducing an aspirator which he had brought from Berlin, aspirating, and injecting the pus into a guinea pig. The pus, when aspirated, might not show tubercle bacilli, but the organisms could be positively demonstrated after the injection into the guinea pig by the examination of the animal's organs several weeks later.

Corrective Rhinoplasty.

DR. LEE COHEN, Baltimore: Where the nose has been partially or completely destroyed by disease or trauma, the individual so disfigured is not only an object of pity, but is not accorded the privileges in the social and commercial world enjoyed by other persons. Since the appearance of Tagliacozzi's work, in 1597, rhinoplasty for the relief of such conditions has been an accepted surgical procedure. For some reason, however, corrective rhinoplasty has not received the attention which it merits. Rhinologists in general have shown but little inclination to develop this line of surgery, although the feasibility and advisability of the work have been proved

by the excellent results obtained by Roe, Joseph, Berens and others for a number of years past. This indifference may be due to the fact that there has been absolutely no systematic treatise on this work until the appearance of Joseph's article in the German Handbuch about a year ago. An operator, however clever, might lack sufficient originality to develop a proper technic along these lines, in the absence of such literature.

Previous to the appearance of Roe's publications only methods calling for skin incisions were described as applicable to external nasal deformities. Credit should always be given to Roe as the originator of the subcutaneous method.

The deformities considered are divided into two classes: (1) Idiopathic or congenital; (2) acquired. To the former belong the overdeveloped nose, hump nose, congenital saddle and pug nose, while to the latter belong all sorts of grotesque alterations in shape and position of the nose caused by fracture or dislocation, and by destruction of the bony or cartilaginous framework from syphilis, tuberculosis and lupus. For practical purposes a further subdivision is generally made into deformities affecting the bony and those affecting the cartilaginous portions, although both are frequently involved at the same time. The various types of deformity have been admirably described by Roe, whose classification seems the most comprehensive.

In all methods of correcting these deformities, the main object should be to avoid marring the subsequent appearance of the nose by scar, or, in other words, to work entirely from within. For these subcutaneous operations incisions, as first advocated by Roe, should be made within the vestibule of the nose, generally above the lower lateral cartilage of the left side, and subsequently, if necessary, on the right side, cutting through the mucous membrane and the cartilage to the under surface of the skin. Whether this incision is made close to the septum or more laterally depends on whether it is desired to alter the line of the nasal dorsum only, or whether the nasal processes are to be severed from their attachment to the superior maxillary. It is surprising to see with what ease one can, with a very small straight knife, undermine the skin over the entire nose through a small incision on one side, owing, of course, to the flexibility of the cartilaginous portion.

By following the course of the knife under the skin with the index finger on the outside, it is not difficult to avoid injury to the skin. The skin having been undermined, one proceeds with the subsequent steps of the operation, working under the skin as if it were a tent, and guiding the movements of the various instruments with the left hand over the outside of the nose. The technic for the correction of the various conditions named is given in detail, and a number of cases reported.

DISCUSSION.

DR. JOHN O. ROE, Rochester, said Dr. Cohen's paper is a verification of the old saying that "imitation is the sincerest flattery."

Continuing, Dr. Roe said: "I am sorry, indeed, to see that Dr. Cohen is inclined to give me scant recognition as the originator of subcutaneous plastic surgery in the correction of nasal deformities, for it must be recognized and distinctly understood that this method of correcting nasal deformities subcutaneously originated entirely with me.

"In describing the method of making the initial incision from the interior of the nose, Dr. Cohen says: 'For these sub-mucous operations, the incision as first advocated by Roe should be made within the vestibule of the nose.' I wish to say that this method was not simply advocated by me but, as a matter of fact, was originated by me. It is my method which Dr. Cohen has copied from start to finish, without the courtesy of giving credit for it.

"Dr. Cohen on one occasion had the privilege of witnessing my work, which I am always glad to show my confreres, and which, as he stated at that time, was the incentive that gave him his special interest in the correction of nasal deformities. The second case that Dr. Cohen mentions in his paper—the case of the congenitally oversized nose, causing the young man so much annoyance and mental distress—Dr. Cohen had the courtesy to refer to me, and came with the patient to my office to see the operation, which I performed at that time in his presence on May 30, 1912. In connection with this operation he saw the distinct method of elevating the skin or 'undermining' it, as he chooses to term it, the forms of dressing necessary in these cases, the use of my saddle splint, which I have used from the beginning of this work,

and the method of holding it in place with the strap of adhesive plaster across the face, the use of the strips or pieces of adhesive plaster under the saddle, one placed above the other, in order to produce extra pressure at points where necessary, the making of the nose narrower by the removal of cartilage and bone, and by fracture, the shortening of the nose by taking out a sufficient amount of the cartilage of the anterior portion of the septum and of the alæ, and stitching the projecting part of the nose back as far as necessary to correct the deformity, and the use of the sling of adhesive plaster to draw up and support the end of the nose after operation. All of these points and others, in the management and technic Dr. Cohen had the opportunity to see and to learn about, for all of them were employed on this patient, whose case was a complicated one. In this case also the nose was altogether too high, and had to be cut down, and the method by which it was done Dr. Cohen has had the discourtesy to give credit to Joseph of Berlin, whereas the same operation was described and illustrated by me in an article on 'The Correction of Angular Deformities of the Nose by a Subcutaneous Operation,' and published in the *Medical Record*, New York, July 18, 1891, which was seven years previous to any published article by Joseph on this subject.

"It would have done Dr. Cohen no harm to have had the courtesy to mention the facts as above stated instead of leading the reader to infer that these methods were largely his own methods and thereby indirectly trying to appropriate them to himself. Joseph of Berlin is also quite in line with the European Germans, many of whom ignore American authors, although quite willing to appropriate their work.

"The technic which Dr. Cohen has collected and described in his paper, however, applies only to the correction of angular deformities of the nose, which he chooses to call 'hump nose,' or to the correction of unduly large or 'oversized' noses, as he designates them, and to deviated noses, the extent to which he has pursued the subject up to the present time. The other varieties of deformities, those attended with depressions and other distortions of the nose, he will find much more difficult."

In emphasizing the fact of having originated the method of correcting nasal deformities by operation subcutaneously per-

formed, Dr. Roe further said: "It does not matter what modifications may be made in the operation in any particular case—whether the operation is done by cutting up or cutting down, with a knife, with bone scissors, with a saw, with a chisel, or with whatever instrument or instruments that can be most advantageously employed in that particular case, the fact remains that the subcutaneous correction of nasal deformities is a method which I have the honor, if honor there be, to have originated and first employed. This fact should not, as a matter of professional ethics, be ignored or appropriated by another without due recognition."

DR. COHEN, in closing the discussion, said he had no intention, in his references to Dr. Roe's work, of detracting therefrom. He insisted, however, that there was no systematic description of this work prior to that mentioned in the paper. He had operated upon about ten cases before he ever saw Dr. Roe operate.

**The Efficacy of Vaccines in the Treatment of
Chronic Diphtheria Carriers.**

DR. ARTHUR I. WEIL, New Orleans: The occasional persistence of the Klebs-Loeffler bacillus in the throats and noses of patients for weeks and sometimes months after their complete clinical recovery, has been a source of annoyance and concern to everyone who has had to deal with diphtheria patients. The necessity for complete isolation of such patients as long as the organisms in virulent form are present, self-evident though it is, entails a serious loss of time and inconvenience to the patient. This is true of not only those carriers who have just recovered from an active diphtheria, but likewise of that other class in whom, though they have never had an active diphtheria, nevertheless, either through contact with diphtheria patients or otherwise, the Klebs-Loeffler bacilli are present. The necessity for isolation is just as great, and the presence of the germ just as persistent, as in those who have actually suffered from the disease.

It is the eradication of the diphtheria bacilli from the nose, throat and ears of both classes of carriers, and the resultant stamping out of a diphtheria epidemic in a public institution, an orphan asylum, that forms the basis of this communication.

After a year or more of sporadic outbreak of the disease,

constantly recurring in spite of the most careful isolation of the diphtheria patients, twenty-four cases finally remained in the isolation wards of the infirmary in which all the active cases had been treated. The patients were classified in three groups: (1) Those who, having had an active diphtheria infection, had been clinically well for a period of two weeks or longer, but in whom live diphtheria bacilli were still found in the cultures. The period during which a positive culture had persisted after complete clinical recovery varied from three months to something over two weeks. There were nine of these patients, who were considered chronic active carriers. (2) Those in whom, though they have never had an active diphtheria, the culture showed the presence of diphtheria bacilli. It is impossible to know just how long they had been carriers, since the bacilli were found in routine examinations of all children in the institution. There were twelve of these, and they were considered chronic passive carriers. (3) Those who had an active diphtheria infection, but in whom less than two weeks had passed from the time of their complete recovery to the beginning of the vaccine treatment. This group comprises three patients, who are called active carriers. Of the three active carriers comprising group 3, in one case ten days and in the other two about one month elapsed between their complete clinical recovery and the last positive culture. Their period of terminal isolation, accordingly, did not seem to be diminished. The cases, however, are too few to allow any definite conclusions to be drawn.

In drawing conclusions as to the value of the vaccine treatment in the twenty-one chronic carriers, the possibility must not be lost sight of that many, if not all, might have cleared up without the use of vaccines. In view of the fact, however, that practically all the cases showed a marked diminution in the number of bacilli present shortly after the treatment was begun, and that all of them eventually did clear up with large doses, the belief would seem to be justified that the vaccines are of some value. At any rate, it has been shown that large doses of diphtheria vaccines can be used without the slightest inconvenience to the patient. From the author's experience, large doses give better results than small ones. Whether autogenous or stock vaccines are the more useful is a matter of opinion.

NEW YORK OTOLOGICAL SOCIETY.

Regular Meeting, Tuesday, May 28, 1914.

THE PRESIDENT, DR. ROBERT LEWIS, PRESIDED.

Mastoid Opened for Relief of Pain.

DR. STEPHEN H. LUTZ reported two cases of mastoid pain, worse at night. The first was a girl of fifteen years, where, after trying various topical and internal remedies, the mastoid was finally opened and the pain relieved. The drum and hearing were normal and no disease was discovered in the bone. The second case resembled the first in most particulars. Both cases were more or less neurotic, but the pain was severe enough in both cases to make the patient miserable from loss of sleep and the long continuance of the trouble. The pain was relieved so rapidly and so completely that Dr. Lutz feels sure, under the same circumstances, he would operate earlier. To save pain for several weeks it is certainly worth trying even as severe a measure as a mastoid operation.

DISCUSSION.

DR. WHITING inquired if the same result could not have been secured by an incision of the periosteum. Dr. Lutz replied that such an incision had been made without permanent results.

DR. BERENS spoke of a similar case in his own practice where, upon opening the mastoid, the picture which he described as one suggesting stalagmites and stalactites was found. The pain in his case was relieved by the operation.

DR. WHITING spoke of a case, at one time under his care, of a woman who had persistent mastoid pain and who had insisted upon the mastoid being opened. Cupping was all that was done. This after a time relieved her of her symptoms. He would not favor opening up the mastoid in the presence of normal hearing and a normal drum.

DR. KERRISON referred to a case where pain of long duration in the mastoid was relieved by Wilde's incision.

Pain, Tinnitus and Deafness With Sensation of Foreign Body for Four Years.

DR. BRYANT reported a case diagnosed by him as otosclerosis. The patient was a student, a woman twenty-eight years of age, who had pain in her ears, tinnitus and deafness, and a sensation of a foreign body in the ears for the last four years. Examination showed nothing abnormal about the middle ears, except blush on the promontories. The eustachian tubes were open. There was a retraction of high and low limits of tone perception, and a prolongation of bone conduction. General treatment of a hygienic character and local treatment to the nasopharynx relieved her of her symptoms.

DISCUSSION.

DR. HASKIN said that he had seen a similar case where the blood examination showed a positive Wassermann.

DR. HARRIS and DR. PHILLIPS questioned the diagnosis.

DR. BRYANT, in reply to Dr. Rae, said there was no fixation of the stapes.

Mastoiditis With Subperiosteal Abscess.

DR. WILLIAM H. HASKIN reported a case of mastoiditis. On January 3, 1912, the nose was operated upon, as a result of which the septum had an oval perforation of one inch by three-fourths of an inch, and was full of hard crusts. Following the operation the patient had O. M. A. P. in the right ear, which discharged continually up to the time of his first visit to the Manhattan Hospital on March 3rd. Dr. Rae at once diagnosed mastoiditis with subperiosteal abscess, and assigned the patient to Dr. Haskin for operation. Cortical perforation was found over the angle of sinus, and as process was badly disintegrated, what was thought to be a complete removal was done. All pain and temperature subsided and wound began to heal rapidly, except that pus escaped from a point just behind the aditus at each subsequent dressing. An autogenous vaccine was made (*staphylococcus aureus*), but in the meanwhile a swelling began below the ear which gradually spread, becoming hard and edematous, until it reached the clavicle. Having begun the vaccine, Dr. Haskin persevered and did not resort to operation until April 13th. He then opened behind the sternoarytenoid muscle and found pus

in large quantity, with a cavity large enough to insert his two fingers, and he could feel the clavicle below and the skull above. Large rubber drainage tubes were inserted above and below through the one opening, which was only about one and one-half inches long. To his surprise, there was very little discharge on the following day. In four days he had removed both tubes, and within fifteen days the wound had completely closed, there being no discharge at any time. The vaccine was given regularly, and to it must be given the credit for this remarkable cure. The mastoid wound healed in four weeks, and on May 10th he had had no pain or swelling, had gained twelve pounds in weight, and the ear appeared normal.

DISCUSSION.

DR. BRAISLIN reported a case of mastoiditis with symptoms referred to both ears, and suggesting a beginning infection of the lateral sinus, in a child of six years. She had been ill with an indefinite ailment for six weeks, having during the latter three weeks lost weight decidedly; had become pale and irritable, and displayed an irregular febrile tendency, her temperature varying from about normal to, occasionally, 101° F. She had been operated on for hypertrophied tonsils and adenoids, two days before the morning when she was first seen by the reporter, and on the day she was first seen she had had a chill followed by a temperature of 104° . It was ascertained that previous to this time she had occasionally complained of slight pain in both ears, but mostly the right. Neither ear had discharged at any time during the illness. Certain isolated lymphatics on both sides of the neck were sufficiently prominent to be recognized without the aid of the fingers. The right mastoid was somewhat tender to pressure, the left not at all so. A diagnosis of right sided mastoiditis was made with presumed involvement of the corresponding sinus. Operation was at once done upon the right side, which showed diseased bone, the knee of the sinus very far forward, so that part of the posterior bony auditory canal had to be cut away in order to reach the antrum. Pus was present throughout the bone and in contact with some firm granulations on the dura, covering the sinus wall. These were not greatly disturbed. The sinus was elastic under pressure, but was rather widely exposed in case symptoms indicated need of opening

it later. The temperature had kept below 100.1° for three weeks subsequent to operation. Six days after operation the glands on both sides of the neck being but slightly if at all diminished, a dose of autogenous streptococcic serum was given, since which the glands are noticeably smaller. Healing of the wound has progressed rapidly.

DR. GRUENING questioned the accuracy of Dr. Haskin's conclusion that the prompt healing of the wound was due to the vaccine.

DR. HASKIN spoke of a second case under the care of Dr. Kerrison, which had been operated upon for mastoiditis. The swelling in the neck had been treated with much benefit by means of the autogenous vaccine, after other treatment had failed.

DR. KERRISON said the case was a baby of thirteen months, who had been operated on by him three months before for an extensive disease of the mastoid. The bone was involved up to and including the portion of the squama, which was quite black. The glands of the neck were much swollen. Two weeks later a radical operation was performed. The post-auricular wound healed only after some time. Later two of the glands broke down and were opened by him and pus evacuated. Dr. Dwyer then took the case for vaccine treatment. The only change in the condition was that instead of a single large mass, the glands were now to be differentiated, the wound behind the ear looked no better. Pus was seen still to be forming.

DR. DUEL referred to the value of the von Pirquet test in children.

DR. HASKIN said this test had not been made in this case.

DR. PHILLIPS said he had reported two similar cases in children, which had proved to be tubercular in nature.

DR. HASKIN, in reply to the question of Dr. Lutz, stated that the infection was of a staphylococcic nature.

DR. BERENS suggested syphilis as a possible cause. He had seen such a case.

DR. GRUENING doubted whether the vaccines were of any value in such cases, and thought we must still proceed to do otology on a clinical and not a laboratory basis.

DR. WHITING expressed surprise at the early destruction of the incus in this case.

The Effect of Salvarsan Upon the Acoustic Nerve.

DR. TOEPLITZ spoke of salvarsan in its effect upon the acoustic nerve, referring to the reports from the Vienna clinics, and his own investigations, which show no neuropathic effect of salvarsan, in contradistinction to those of the Vienna clinic, the many disturbances of the acoustic nerve of which stand alone in this respect.

DISCUSSION.

DR. DUEL said that two meetings before, he had presented a patient where deafness had followed the use of salvarsan, and that he had at that time gone to some length into the statistics quoted by Dr. Toeplitz.

DR. DENCH spoke of a case of syphilis where the injection of salvarsan had been followed by deafness in one ear. He gave as his opinion in this case, that the deafness was due to an insufficient dose, and had advised a repetition of the injection. He promised to report the result at a subsequent meeting. He referred to the paper by Dr. Fordyce, read before the Section on the Eye, Ear, Nose and Throat, at the recent meeting of the Medical Society of the State of New York, in Albany, in which he advocated perfected technic as a means of preventing untoward results.

Supplementary Report in a Case of Total Deafness.

DR. MCKERNON made a supplementary report in a case of total deafness reported at the March meeting. The patient, a boy, had suddenly become totally deaf. Examination showed the drum membranes black in color. There was a history of suppuration from the ears two years before. The Wassermann reaction was negative. The boy was put to bed and given hypodermic injections of pilocarpin in from one-sixth to one-half grain t. i. d., and strychnin, one-fiftieth to one-fifteenth grain. In four weeks' time the hearing was restored. In six weeks the drum membranes became normal. The vertigo complained of disappeared at the end of the second week. There was no moisture in the middle ear.

DISCUSSION.

DR. LUTZ inquired as to the presence of a clot in the middle ear.

DR. MCKERNON replied that he did not think any clot was present.

DR. DENCH spoke of an old gentleman who had come to him absolutely deaf in one ear, where the hearing was restored in inflation.

DR. HASKIN raised the question of hysteria.

DR. MCKERNON replied that two neurologists had seen the boy and had carefully examined him with this in view, and both had positively excluded it.

Pain in the Ear and Over the Mastoid in Conjunction With Appendicitis.

DR. PHILLIP D. KERRISON spoke of a case that he had seen recently in consultation. The patient had been operated upon for appendicitis and was complaining of pain in the ear and over the mastoid. There was a history of an old middle ear suppuration. At the present time there was no discharge from the ear. There had been pain in the mastoid before. He had given the opinion that the pain was not due to any inflammatory disease in the mastoid.

DISCUSSION.

DR. PHILLIPS thought it might be a case of eburnating mastoid.

DR. DENCH said that he had seen a girl with pain in both mastoids with normal drum membranes. The X-ray had shown a displaced wisdom tooth on each side. In another case there was pain in the mastoid, and examination had shown a cavity in a tooth. In both cases the pain was cured by attention to the teeth.

Labyrinthitis Following Mumps.

DR. WENDELL C. PHILLIPS reported a case of labyrinthitis in a boy of eighteen following mumps. There had been vertigo and a high temperature for two weeks. He was totally deaf in one ear. For a time he was partially unconscious. The hearing had not improved in the affected ear.

DISCUSSION.

DR. PAGE inquired whether any gentlemen present had seen the hearing clear up when affected by mumps.

DR. McKERNON and Dr. Dench said that they had seen cases where it had improved, but no cases where it had become entirely well.

Meningitis Following Fracture of Skull.

DR. EAGLETON reported two cases of meningitis, the first following fracture of the skull; the second, probably tubercular in nature. He had operated according to the method recommended recently by Dr. Haynes, and could confirm Dr. Haynes' statement that the cerebellum would in this operation not prolapse into the wound. The usual rigidity following operation on the brain is not seen following this operation. Both patients were alive, and although they probably will die, he felt that the operation was a decided advance in brain surgery.

DISCUSSION.

DR. DENCH said that he had recently operated on a case by the Haynes method which was still alive. There was considerable rigidity. He said that the operation was not a new one. It was described a number of years ago in Cunningham's "Anatomy."

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL
SOCIETY.

Regular Meeting, April 21, 1914.

DR. OTTO J. STEIN, THE PRESIDENT, IN THE CHAIR.

Case of Singer's Nodule on the Vocal Cord.

DR. GEORGE E. SHAMBAUGH presented a patient from whom he had successfully removed a singer's nodule from the vocal cord. The patient was a clergyman, and had been troubled with hoarseness for about three months, which interfered very much with his speaking voice. The nodule was about the size of a pinhead and was located on the usual spot for these nodules, that is, at the junction of the anterior with the middle third of the vocal cord.

Dr. Shambaugh showed the instrument which he has used in several cases to remove these nodes. The ordinary forceps are entirely too clumsy for this work, and the use of cauterizing substances is obviously dangerous.

Such nodules often develop rather quickly, as illustrated by a public speaker whose voice became affected during a reelection campaign, and in another case cited, where the defect had followed the excessive use of the voice during a football game.

There is a tendency for the nodes to disappear spontaneously, but very often they will persist indefinitely until removed in an operative way.

Case of Frontal Sinusitis and Ethmoiditis With an External Fistula.

DR. SHAMBAUGH also showed a case of a young lady who was referred to him because of a fistula about one-quarter of an inch above the inner canthus of the eye, which had existed for seven weeks. The development of the fistula followed six weeks of severe headache, which had been treated as a frontal neuralgia. She gave as the cause of her trouble an accidental kick from a child's shoe six weeks before her headache developed. The maxillary sinus on that side was

quite dark. Both frontal sinuses were small, as was shown by the X-ray plate, and the transillumination, for this reason, gave very little difference on the two sides. A removal of the middle turbinate body and exenteration of the ethmoid labyrinth gave a free passage into the frontal sinus. The discharge from the fistula ceased immediately after the operation, and the case went on rapidly to complete recovery.

DISCUSSION.

DR. E. L. KENYON wished to speak especially concerning the case of apparent singer's nodule mentioned by Dr. Shambaugh as coming on after scarlet fever. Ordinarily, singer's nodules are dependent upon misuse or overuse of the voice. In the case referred to a nodule appeared following scarlet fever, on the cord, but in the usual place of the singer's node. There may be important significance in this, because many years ago Fränkel discovered a little gland on the cord, not regularly, but at times, which was situated between the middle and anterior third of the cord. Later, Imhofer, of Prague, in three cases of singer's nodes saw a secretion coming out from a gland in this particular region, thus confirming Fränkel's discovery. It seems that singer's nodes from this latter glandular cause are in contrast to the more usual non-inflammatory type, accompanied by a local inflammation. The local inflammation produces an inflammation of the gland, hypersecretion of the structure, and by the closing up of the duct leading outwards, a swelling on the cord is produced. That is a very important fact to know, from the standpoint of the singer. Many of the people with these nodes have voices worth many thousands of dollars, and they must be handled with extreme care. If you cauterize a node due to such a glandular inflammation, you are almost certain to prevent recovery, because you are liable to close up the duct of the gland. If you operate, you are very likely to do the same thing. Clinically, it seemed to Dr. Kenyon, that he has never seen one, to know that it was of this type, but it is stated that this particular form of singer's node has a flattish contour, and, clinically, that it almost always gets well simply by rest of the voice. The speaker thought it well to bear that fact in mind, since the case mentioned had followed a disease which could easily have produced inflammation of the vocal cords.

One must bear in mind that singer's nodes may recur, because the condition is due to misuse of the voice. The important thing is to correct the manner of handling the voice, so that the cause for the formation of the nodes shall be done away with.

DR. OTIS H. MACLAY was interested in the frontal sinus case especially, owing to the fact that it was a chronic case and was cured by intranasal work. This case would indicate what he always believed, namely, that in these cases of frontal sinus trouble it is well to do all we can intranasally before undertaking the external operation. He reported a case seen by him in which there was a large mass of pus in the forehead and a denuded area of bone. There was no external opening, but you could feel when the pus was relieved that there was bone necrosis. Good drainage was obtained through the nose. A small incision was made in the skin under gas; the pus poured out. In a couple of days there was no drainage from the external wound. The intranasal work consisted of removal of the anterior portion of the middle turbinate and the anterior ethmoid cells, and a liberal enlargement of the nasofrontal duct.

DR. JOSEPH C. BECK said that he would interpret the X-ray picture shown by Dr. Shambaugh as indicating an ethmoiditis. It is very easily shown that the ethmoidal cells and not the frontal are involved. Removal of the turbinate and cleaning out the ethmoid would clear up the condition.

In reference to the singer's nodes, he has recently removed two by means of the suspension method, which has proven satisfactory in his hands. These nodes are very easily removed with an instrument similar to that described by Dr. Kenyon. With such an instrument you just get the edge of the node, and it is impossible to go any deeper.

DR. CHARLES H. LONG asked Dr. Shambaugh, or anyone else who had had experience with these growths, if they had ever used fulguration?

DR. O. J. STEIN said that he had used fulguration, with no satisfaction.

DR. R. H. BROWN cited the case of a man, seventy-five years of age, who had a sinus affection similar to that in the case reported by Dr. Shambaugh. This had been discharging externally for some time. He was rather old for extensive

operation; however, some of the middle turbinate was taken away. A large polyp was found and removed, leaving the ethmoid cell from which it grew. This was followed by a good result. It was plainly a case of anterior ethmoidal cell disease. The discharge continued internally, but never broke externally again during the five remaining years of life.

DR. J. R. FLETCHER cited a frontal sinus case. About two years ago a lawyer was sent to him with an orbital abscess. The pus was coming from the upper eyelid. He had a history of having swelling there three or four times before, and had been under treatment for about six months. The speaker washed the frontal sinus, and asked for an X-ray picture, but the man did not seem to think it necessary. Finally, with a probe entering here (indicating), it went in such a short distance that he could not understand it. He probed into the frontal sinus, and could not meet the end of the other probe. He started again and went in for a short distance and met it. So he came to the conclusion that he had an ethmoidal bulla to deal with. He got absolutely nothing out of the frontal sinus two or three times. He could go a relatively short distance, and by bringing the probe out and going just a little behind it, he could go a much greater distance. He then washed that out with simple bluing to see whether there was actual communication, or whether he had deceived himself by finding denuded bone. The probe was a cannula. Through that he passed this ordinary bluing water, and it came out through the nose. The anterior end of the middle turbinate had been removed before he came to the speaker. He simply mentioned these bullæ, because we must remember the two kinds—the ethmoidal bulla, which is attached to the frontal plate, and the bulla frontalis, which is attached to the cranial plate.

DR. SHAMBAUGH, in closing, stated that the instrument with which he had removed the nodes from the vocal cords was small enough to be placed between the vocal cords, as suggested in the remarks by Dr. Kenyon.

As to the question whether in the second case presented there was any frontal sinus disease, Dr. Shambaugh pointed out that because of the small sinus, the X-ray plate and transillumination were deceptive, since neither gave any clear indication of frontal sinus trouble, and yet on introducing a cannula and irrigation, pus was washed out of the frontal sinus.

Brain Cyst.

DR. H. KAHN presented a case of brain cyst, which had been operated, and presented two points of interest: First, the long standing period of the disease; second, the help of the X-ray in diagnosing the condition. This latter point was in opposition to a statement once made by Dr. Beck, that he never had any help from the X-ray. Dr. Kahn did not know whether the X-ray was absolutely conclusive or not, but it was of great assistance in this case. The man was twenty-five years old. When nine years of age, while driving, he struck his head and apparently suffered from a fracture of the skull. He was operated for this, so he says, but no evidence of such operation could be found at the present time by the speaker. From then up to the time when first seen the man had several attacks of otitis media, for which he was treated. He came to the hospital about the 3rd of December, complaining of excruciating pains in the ear and right-sided headache, and was admitted to Dr. Kahn's service. This headache was definitely located on the right side, from the back of the neck to between the eyes. Very sensitive to touch and pressure. At that time temperature was normal, pulse 72 to 76. The ear showed a small pin point opening in the inferior posterior quadrant, with pus pulsating under pressure. A paracentesis was performed, which did not seem to relieve the symptoms. The headache and pain both continued. In addition, he had a nystagmus to both sides. On washing out the ear with hot water the nystagmus was uninfluenced; cold water gave a very strong and pronounced nystagmus to the left. He was kept under observation for four or five days, when suddenly one day, while in bed, he became very cyanotic and very dyspneic; the pulse became weak and thready for a little while. When he came out of this attack the headache was very intense on the right side. He had three or four similar attacks. Two X-ray plates were made, with hope of finding what was in the mastoid, which was thought might be at fault. The frontal view showed a sclerosis of the mastoid above and a line below, showing a large terminal cell and a dark area in the brain shadow. Those findings were verified at operation. Dr. Fletcher came in consultation at that time, and pointed out this mastoid cell. The second plate was rather difficult to read. It was taken at an angle, showing the mastoid with a black area above. A few days after these pictures were

taken a radical mastoid was performed, showing the condition of the mastoid, as read in the picture by Dr. Fletcher. The brain was exposed and a needle was inserted in an upward forward inward direction, as indicated by the dark area of the X-ray plate, and a syringe of thin fluid was withdrawn which on culture was found negative after six days, showing no organisms at all. Differential count showed thirty-six polymorphonuclears and fourteen mononuclears; no Gram positive organisms shown. The wound was left open, and in a couple of weeks it was healed up. It was not thought wise to open the cyst at the time of the mastoid operation, for fear of infecting the brain. At the end of a month the patient returned with the same symptoms of headache, nystagmus, and so forth. After three or four days in the hospital a lateral opening was made, the bone was drawn back, showing absolutely no pulsation of the brain. A needle was inserted, yellowish brown fluid was withdrawn, and immediately pulsation of the brain followed. On slipping back the dura the vessels of the brain were found very large and varicose. It was impossible to make an incision into the brain without great hemorrhage. A trapdoor opening was made, about one inch by two inches, and in spite of hemorrhage the speaker introduced his finger and found the cyst to be about three by two and one-half inches in size, apparently multilocular, with a large multilocular area above. Hemorrhage was very great, so the wound was firmly packed, leaving a strip of vioform gauze for drainage. The packing was left in for twenty-four hours, when it was removed gradually, and at the end of forty-eight hours it was all out. Apparently, the patient is cured—at least, up to the present time. Whether the cyst will refill or not, the speaker could not say. All the symptoms disappeared except a slight nystagmus to the right. The other symptoms have all disappeared entirely, and the patient appears to be in good health now.

The speaker forgot to say that the tuning fork showed the Weber to the left—the good ear—high tones apparently not heard; Schwabacher not heard; somewhat shorter Rinné.

DISCUSSION.

DR. J. R. FLETCHER said the interesting point about the case to him was that it was a middle fossa abscess, which Dr. Kahn had omitted mentioning. In spite of that, the man

had a nystagmus. The nystagmus was attributed to the size of the cyst, making indirect pressure upon the cerebellum. The patient had an exaggerated patellar reflex on the same side. Another important thing was that the man had had several of these attacks previously, consisting of lowering of respirations, lowering of temperature and subnormal and lowered pulse. When Dr. Fletcher first saw him the pulse was 85, respirations 14, and temperature 97°, which was practically normal. One other interesting feature about the case was that it was possible to make a diagnosis of latent abscess. Nothing could be seen. The speaker's idea was that sixteen years before, this man had had an abscess in the middle fossa, and that it was at present in the period of latency; that these attacks were explained on the basis of hemorrhage into the abscess, or into the area of the cyst—which it turned out to be. Blood was found in the first puncture.

DR. J. HOLINGER asked if there was any pus shown at any time, or was the contents of the cyst or abscess not simply cerebrospinal fluid mixed with the blood? The whole history seemed to him to be somewhat similar to one that he was much interested in. He had made a diagnosis of brain tumor from the tuning fork findings four years ago. The patient had been getting along very nicely for four years, after a decompression operation, but again had attacks of dizziness, severe headache, and her sight and hearing grew worse. A second operation had to be performed. The same flap as that used at the first operation was formed. The intracranial pressure was less than at the first operation. As soon as the flap was down, a cyst the size of a hen's egg showed, and a large amount of cerebrospinal fluid escaped. The cyst opened and it was evident then that it was the posterior horn of an enlarged lateral ventricle. The fact that Dr. Kahn's cyst was drained towards the ear does not speak against the possibility that it might have been the posterior horn. This is important on account of the therapy of the case. If this is an internal hydrocephalus, it will fill up again; it will have to be permanently drained, by inserting a small silver drain from the cyst to underneath the outer skin. The cerebrospinal fluid, which is produced in too large quantities, causes these enlargements. It will be led outside of the skull cavity and will be absorbed in the tissue outside of the skull, and intracranial pressure cannot rise again.

DR. JULIUS GRINKER, on invitation, simply wanted to ask a few questions. First, was a lumbar puncture made, and, if so, what was the finding? Second, he would like to have an exact description, topographically speaking, of where the cyst was really found and opened. Third, is this not, after all, a meningitis serosa circumscripta, a condition which had been frequently described in connection with inflammatory conditions of the ear?

DR. JOSEPH C. BECK wanted to say just a word with reference to Dr. Kahn's statement that he (Dr. Beck) had said that X-rays in mastoid cases were of no value. He believes a great deal in them. However, the round cell that was pointed out to him was, in his opinion, a broken-down mastoid cell, in contradistinction to the opposite side, which showed a number of cells. With reference to diagnosis of brain conditions, it was in such cases that he said that he had yet to see a plate that showed anything that could be proved by operation as an abscess. He could not see that the plates passed around by Dr. Kahn showed anything definitely. Dr. Beck is of the opinion that X-ray pictures show best when stereoscopically taken.

DR. KAHN, in closing, said that he thought Dr. Beck misunderstood him. He had understood Dr. Beck to say at a meeting that he had never been helped by X-ray pictures in brain conditions. He (Dr. Kahn) had been helped somewhat, and especially in this case, because he had found the cyst in the place where the dark shadow apparently was in the plate. Of course, this was only one case.

In answer to Dr. Holinger, no pus was found in this fluid at all. It might be, of course, a lateral ventricle diverticulum, but there was the fact to be considered that it apparently was a secondary result of the traumatism years before. One thing could be assumed as well as another. The cyst may fill up again, but it would be necessary to wait to see whether it did or not. The case was presented merely to show that it had been operated, without apparent success.

DR. HOLINGER said that so long as the wound is still open it would be a good plan to insert a drain.

DR. KAHN said he was very glad of the suggestion, and would do it.

Replying to Dr. Grinker's question: A lumbar puncture

was made and was negative. As to the convolutions, he could not say. The cyst was opened, incision being made directly over the external auditory meatus, and so far as the convolutions of the brain at the time were concerned, it was apparently impossible to tell exactly, on account of the varicosity of the vessels and the apparent edema about them. He did not think it could have been a circumscribed meningitis, because it was too deeply situated within the brain structure. At operation he went through an inch, approximately, into the brain structure. It was always beneath the meninges, and so he thought it would not have been of meningeal character, and must have been in the brain.

Case of Simple Mastoid Operation Under Local Anesthesia.

DR. GEORGE W. BOOT presented a little girl who had had mastoiditis, but was in too bad a condition to be given a general anesthetic, and it was necessary to operate under local anesthesia, namely, one per cent novocain. She stood the operation very well, made very little complaint of pain—only when going through the soft tissues.

Thrombosis of Superior Longitudinal Sinus.

Dr. Boot also showed a specimen from a young woman who was taken sick with symptoms pointing toward suppurative otitis media.

M. H., female, aged twenty-two years, single, university student. Admitted to the German Hospital on June 3, 1911, in an unconscious condition. Patient had a cystitis on admission, for which she received boric acid irrigations of the bladder. Her temperature was 102°; pulse, 80; respirations, 22. On June 4th the patient was restless, crying loudly and tossing about. She slept at intervals. Part of the time her limbs were rigid and the back of the neck painful. Temperature ranged from 101.6° to 103°; pulse, 84 to 108; respirations, 24. Blood examination gives ninety per cent hemoglobin by the Talquist scale. Whites, 14,680; reds, 4,500,000. Urinalysis gives color dark straw. Transparency clear; a deposit of heavy sediment. Specific gravity, 1022. Reaction alkaline. One hyaline cast found. Numerous epithelial cells present. Thirty-two pus corpuscles present in field of one-sixth ob-

jective. Fourteen blood corpuscles present in field of one-sixth objective. Bacteria present; spermatozoa absent; albumin present in moderate amount; sugar, none; calcium oxalate crystals numerous; yeast fungus present.

June 5th: Patient very restless; crying; biting lips at times. At 5 p. m. patient had severe convulsion; had urinated involuntarily ever since admission; menstruating. At 12 p. m. very restless; crying; throwing left arm and leg continuously. Temperature ranging from 102° to 102.4° ; pulse, 80 to 120; respirations, 22 to 26. On June 6th urinalysis showed: Color, amber; reaction, acid; no casts; five to six epithelial cells in one-sixth objective; twenty-five to thirty pus cells in a field of one-sixth objective; a few bacteria; albumin present in moderate amount; sugar, none; a few calcium oxalate crystals are present; triple phosphates numerous; no amorphous deposits. At 1 a. m. patient very restless; slept about one and one-half hours during the night; drank about twenty ounces of water. At 5:36 a. m., convulsion lasting nine minutes; right eyelid twitched, then right arm and head, then leg. Left part of body remained rigid. Right arm twitched for three minutes after rest of body stopped. At 8:33 a. m., convulsion lasting thirteen minutes. At 10:33 a. m., convulsion lasting twenty-one minutes. Left side of body quiet. At 4 p. m., lumbar puncture followed by convulsion. Patient very restless. At 7:38 p. m., slight convulsion, lasting six minutes. At 10:30 p. m., convulsion lasting eighteen minutes. Patient hiccupped during entire convulsion. Temperature on this day ranged from 101° to 104° ; pulse, 80; respirations, 80 to 136.

June 7th: 2:15 a. m., convulsion lasting seven minutes and starting in right arm. 3:10 a. m., convulsion lasting four minutes and starting in right arm. Some hiccup. 4:12 a. m., convulsion. 6:13 a. m., convulsion lasting eighteen minutes; cried before and moaned during the convulsion. 10:03 a. m., convulsion lasting seven minutes. 11:10 a. m., convulsion lasting eight minutes.

Dr. Boot saw her about noon and found the drum membranes were not markedly reddened, but slightly so, and gave the appearance of those seen in a middle ear catarrh. Accordingly he did paracentesis. The trouble in this case was very obscure. The patient could not talk. She would look in the

direction of those in the room, but gave no sign of comprehending anything said, and made no reply.

1:30 p. m., convulsion lasting twenty minutes. 4:30 p. m., convulsion lasting nine minutes. 6 p. m., convulsion lasting four minutes. 8:10 p. m., convulsion lasting eight minutes. 8:20 p. m., twitching, not severe; 9:30 p. m., convulsion lasting eight minutes. Temperature on this day had varied from 101° to 102.4° ; pulse, from 100 to 156; respirations, from 20 to 26.

June 8th: 1:30 a. m., convulsion lasting eight minutes. 4 a. m., convulsion lasting nine minutes, during which she screamed continually. 5 a. m., convulsion lasting four minutes. 7 a. m., convulsion lasting four minutes; very restless and screaming at times. 1 p. m., convulsion lasting five minutes. 4:15 p. m., convulsion lasting nine minutes. 7:30 p. m., convulsion lasting four minutes. 10 p. m., convulsion lasting four minutes. Tossing of arms and legs continually. Temperature varied from 100.6° to 102.2° . Pulse varied from 100 to 158. Respirations varied from 22 to 28.

June 9th: 6:30 p. m., convulsion lasting six minutes. Convulsive attacks every few minutes. Noon, convulsion for three minutes. 2 p. m., slight convulsion. 9:55 p. m., death.

Postmortem Notes.—June 10, 1911: Body of a well developed young woman, said to be twenty-two years of age. Nothing abnormal was noted externally. Only the head was opened. Before the dura was opened, the brain seemed to be unusually dark. On removal of the dura, the sinuses of the vertex, the superior longitudinal sinus, the lateral sinuses as far as the mastoid on the right side, and throughout on the left side, and the torcular, were all thrombotic, as were also all the veins of the meninges of the vertex. The thrombosis and congestion were most marked on each side of the superior longitudinal fissure, and on the left side along the fissure of Rolando. About the middle of this fissure on the left side was a small amount of yellowish exudate in the meshes of the pia. Traces of a similar exudate were seen here and there lying alongside of large thrombosed veins. Most of the brain was firm, but lying parallel to the superior longitudinal fissure was an area about two by five centimeters that felt distinctly soft, as if it were a cavity filled with liquid. There was no evidence of disease of the mastoid. The foramen for the

mastoid vein was larger than normal—about four to five millimeters in diameter. The base of the brain showed no thrombosis or exudate of any sort. After hardening for several days in a ten per cent solution of formalin the brain was cut in horizontal sections. The right hemisphere showed a marked area of congestion in the frontal lobe. The left hemisphere was softened throughout the frontal and parietal lobes. The congestion was most marked in the region of the insula, and the softening most marked about the center of the white matter. The softened area showed numerous points of congested vessels. The area not congested was a greenish yellow color, and appeared as if on the point of breaking down into pus. All the larger veins were thrombotic. The brain and dura with the thrombosed sinuses were put into ten per cent formalin solution for further examination. When the brain was sectioned after hardening it was found that there was a large area of softening in the left hemisphere and a smaller area in the right. It appeared as if had the patient lived a few days longer, the whole left hemisphere would have become one large abscess cavity.

DISCUSSION.

DR. ROBERTSON asked the speaker if he thought the trouble came from the ear or nose.

DR. BOOT replied that, so far as he could find out, it came from neither of them.

DR. SHAMBAUGH asked if there was the fluctuating temperature of a sinus thrombosis.

DR. BOOT replied that there were no sudden falls of temperature; no chills. The only thing to point to the source of the infection was an old cystitis.

DR. C. M. ROBERTSON said that longitudinal sinus thrombosis is one of the rare things found in inflammatory conditions in the brain. He thought that in this case it was probably a postmortem clot occurring near the end of the life of the patient. If she had a thrombosis of the longitudinal sinus she would certainly have been in worse condition than if she had had thrombosis of the lateral sinus, and the lateral sinus on that side would probably be thrombosed anyhow. There have been a few cases of longitudinal sinus thrombosis, but

they are decidedly rare, and nearly all follow infection about the region of the cribriform.

DR. JULIUS GRINKER said that most of the cases of longitudinal sinus thrombosis have been diagnosed in the same way as that of Dr. Boot—that is, postmortem.

DR. BOOT, in closing, and replying to Dr. Robertson, said that the changes in the brain, the softening and the hemorrhages, pointed to the thing being an antemortem affair—not a postmortem clot.

The Speech Aspects of a Case of Cleft Palate.

DR. ELMER L. KENYON presented a patient, Mr. E. T. B., a young man of twenty-four years, who represented the less complex speech problems of cleft palate. The lip cleft was closed in infancy, but repeated operations on the palate itself between five and seven years were unsuccessful. Not until two years ago was the palatal cleft closed, these later operations—but not the earlier ones—being done by Dr. T. W. Brophy, to whom the speaker was indebted for the patient. Originally, the palatal cleft did not extend backwards completely through the posterior border of the soft palate, but the partly intact soft palate was so short as to permit the tip of the tongue to be easily passed backwards and upwards into the nasopharynx. After closing the bony cleft the problem consisted in lengthening the soft palate. This was done by utilizing both palatopharyngei muscles and the left palatoglossus. The result was a long intact palate, flexible, and distant at the lower border about three-quarters of an inch from the posterior wall of the pharynx. The levator palati muscles take hold well on phonation, but so high above the lower border of the newly constructed part of the palate as to make the application of their power in raising the palate of little effect on the newly constructed lower part. But the power demanded for raising the lower part of the palate is much increased by the relative tightness of the lower border. Consequently, the distance of the newly made lower part of the palate to the posterior wall is not reduced on phonation.

The operative result attained was much more than one could possibly hope for, considering the shortness of the orig-

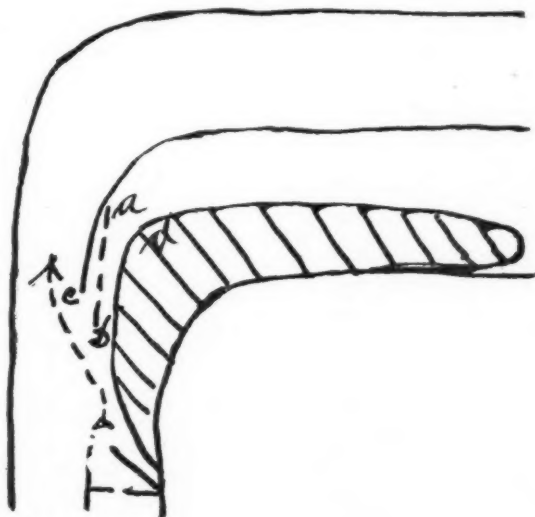
inal palate, and its distance from the posterior wall. The point to be raised later concerning the result was raised not critically, but only in the hope of throwing a little additional light on the speech aspect of the surgical problem involved.

Commenting on the speech of the patient, it should first be remarked that a deflection of the nasal septum, together with chronic vasomotor enlargements of the lower turbinates, produced some rhinolalia clausa. The septum was therefore resected, without, however, disturbing the vasomotor swelling, which still continues and which will not be disturbed. The patient habitually talked rapidly and with a high pitch, largely in the head register, and with habitual lack of clearness. By persistent effort he now talks usually in the chest register and more slowly and distinctly; and certain minor imperfections of articulation have been corrected. But while the speech is markedly improved, the voice, of course, remains nasal, though less so than formerly. There is no hope of producing movement in the lower part of the soft palate, even with the massage being carried out, owing to the conditions previously mentioned.

The question the speaker wished to raise was in reference to the distance the soft palate had best be built downward in such cases, providing it be operatively possible to stop at any point desired. The downward extension of the palate should be such as not to discourage action of the levator palati muscles, and failing in securing such action, the palate should be only so low as should encourage, so far as possible, the outgoing vocal breath to take the oral route of exit rather than the nasal. This point should be determined with reference to the position of the posterior part of the tongue in the production of those vowels likely with an open palate to be most nasal, particularly as in *boot*. In these sounds the posterior part of the tongue raises to a median position in the mouth close to the immobile soft palate. If the lower border of the palate lies opposite the position of the arch of the posterior part of the tongue when the above sounds are produced, the air will be directed for the most part into the nose; whereas, if the lower border of the palate ends slightly above the center of this arch, the vocal breath can more readily pass into the mouth; a too low position of the palate tends, in other words,

to produce a funnel-like connection of the laryngopharynx and the oropharynx behind the immobile palate, and a slightly higher position tends to discourage such a tendency. But if the palate as it descends tends quite sharply backwards, the increased length has no disadvantage of this kind.

The roughness of the anterior surface of this palate, due to the building of the new palate and to the remnants of tonsillar crypts which now enter into the new palatal structure, have a pathologic importance. Particles of food and infec-



tious materials find their way easily into these irregularities, and the patient is, in fact, subject to infections in this region.

The speaker then drew a hypothetical case: If the palate in the accompanying illustration had stopped at a, the funneling into the nasopharynx would have been lessened and the levator palati muscles would take hold more effectively. The palate, a to c, would, however, have less tendency to funneling. Training should aim especially to reduce the distance, a to d, by causing the tongue to be habitually held lower on phonation.

Case of Injured Palate, With Stammering.

The second patient, E. M., a boy of ten years, presented certain questions having reference, on the one hand, to an injured soft palate, and, on the other, to difficulty in nasal breathing with consequent rhinolalia clausa, and also to stammering. There is no important history in the case until at six years of age he began to stammer. Shortly after this an operation was done on the throat, resulting, the mother states, in reducing the size of the tonsils. The mother says that two years later (that is, two years ago) the throat was operated again, this time for adenoids. She says that two unsuccessful operations were done without an anesthetic, and then a few days later a third one with general anesthesia. The stammering was not influenced by these operations. He is under treatment now in the Chicago public schools for his stammering, and his instructor, Miss Powers, who brings him to Dr. Kenyon, states that he is not responding well to training treatment. He breathes much of the time with his mouth open, and when the mouth is closed he has a nasopharyngeal or nasal noise, indicating obstruction, and is usually restless at night. The ears seem not to be involved at present. Examination shows the following: On each side the remnant of the tonsil and both anterior and posterior pillars appear to be adherent into one agglutinated mass. The palate is firmly bound down below, and the distance of the lower border to the posterior wall is not shortened on phonation, although the levator palati take hold well above. The uvula is absent; a heavy linear scar passes horizontally in the soft palate for an inch on the left side near the lower border, and the lower part of the palate seems drawn over towards the left side. The distance from the immobile lower part of the palate to the posterior wall of the pharynx is about one-half inch. When examined, rhinolalia aperta was absent. Finger examination of the nasopharynx shows adenoids to be present sufficient to interfere decidedly with breathing. The nose shows chronic vasomotor swelling of moderate degree.

The first question to be considered had reference to the obtaining of normal breathing by removal of the adenoids. If the adenoids were to be removed, the patient's voice would probably become markedly nasal, exactly as the voice of the

operated cleft palate patient is nasal. The adenoids serve for voice purposes the part of the injured palate. The speaker, therefore, advised against operation for adenoids at this time, but should advise conservative nasal cauterization, which he hoped might do away with acuteness of the difficulty in breathing. If this failed, he should advise removal of small bits of the adenoids, working through the nose with a nasal adenoid forcep, guided by the finger in the nasopharynx. The possibility of operation on the soft palate to obtain movement in the lower part would be considered. If all of this worked out successfully, the patient would always be free from an open nasal voice; in any case, correct voice habits would have been thoroughly established before the nasalizing had begun, and thus its effects mitigated.

Concerning the dependence of stammering upon the condition of the palate or the nasal occlusion: Stammering is dependent fundamentally on psychologic causes. Only where local conditions are capable of producing or increasing these mental disturbances can such local conditions affect the disorder. That adenoids may do so, he is convinced. That such an immobile palate might do so by interfering to a certain degree with the confidence of the patient in the handling of his voice is conceivable. But in view of the distinctly secondary influence of the adenoids and of the immobile palate on the stammering, and in view of the statements above with reference to rhinolalia aperta, the treatment of the stammering should be carried on with unusual care, without local operative measures upon the throat at this time.

DISCUSSION.

DR. CHARLES M. ROBERTSON said that a couple of years ago Dr. Beck showed a case of a lady patient in whom he had injected paraffin to build up the posterior wall, and he also had done that in a case. The speech in the first case exhibited by Dr. Kenyon could perhaps be improved considerably if the wall were built up, so that the palate could meet the lump made by the paraffin, especially in the U sounds, in which the palate should strike the pharyngeal wall. The only difficulty in introducing the paraffin is to get it to stick. Nearly all of the cases operated for cleft palate have an inelastic soft palate

resulting. If the paraffin slips, you can wait a little while and do it over again, doing half the pharynx at a time, and using the paraffin cold. All you have to look out for is to keep it from going up too high into the nasopharynx.

In the second case, Dr. Robertson could not understand how Dr. Kenyon was going to improve the child's speech by leaving the adenoids. He thought Dr. Kenyon was counting too much on the adenoid as a foreign body, because it probably would disappear in a couple of years. The child was just about reaching his adolescent period, when the adenoid should atrophy.

DR. JOSEPH C. BECK advised that there are better methods than paraffin in this work. Since using it in the patient referred to by Dr. Robertson, he had come to this conclusion. While improved temporarily, the paraffin sagged, and the nasal tone returned. He had operated on a second patient, and instead of using the muscles of the palatopharyngeus, the pillars, so to speak, he lengthened the palate, according to Gussenbauer's method, first described in 1886, by severing the soft palate from the hard, taking a thin ridge of palatal bone, making the incision transversely and then uniting longitudinally. Some tension incisions are necessary laterally. That is a far better method than the proposition shown here in the case presented.

DR. MACLAY asked how much was chiseled away.

DR. BECK replied, just the edge to get support for the soft palate, so that it would not drop.

DR. ROBERT SONNENSCHN said that Eckstein, of Berlin, used paraffin in such cases as those reported. As a matter of fact, if the paraffin is used cold, as suggested by Dr. Robertson, you have to use a soft paraffin, which is liable to be displaced in the tissues, whereas if the hard paraffin of Eckstein were used, the substance would remain in situ.

DR. BECK said that the paraffin he used was Eckstein's paraffin.

DR. KENYON, in answering Dr. Robertson, said that if the adenoids remained in the boy shown, the speech habits would be developed correctly, but if they were taken out speech would be incorrectly developed. He did not think of using paraffin, because by this method the speech would be made too excellent.

Herpes Zoster Oticus, With Facial Paralysis.

DR. JOSEPH C. BECK showed a case which he had treated several years ago for peritonsillar abscess, which reappeared at various times. The patient developed a pain in the region of the forehead and along the ear, which kept him awake for several hours of the night, stopping exactly at four o'clock in the morning. Then he developed a slight attack of tonsillitis. The following day he had a similar condition of pain, at precisely the same hour, and stopping at the same hour. On examination, nothing was found about the throat; nothing wrong with the ear or teeth. Closer examination showed a little red spot in the floor of the auditory meatus, externally, which the speaker thought was due to a slight trauma. He told the patient to return the next day, but two or three hours later he called him over the phone and asked him to come and see him, that the water would run out of his mouth when he drank. As soon as Dr. Beck saw the patient he recognized that he had a complete facial paralysis. The next morning he had the beginning of a typical herpes zoster oticus. The next day the whole ear was swollen. This was supposed to be an inflammatory condition. The diagnosis is not at all difficult. One thing he wished the members would speak about in discussion was the etiology. The patient would always have a peritonsillar abscess when he had these symptoms of throat trouble, but with this attack he did not have the peritonsillar abscess. Was this peritonsillar abscess possibly an etiologic factor in most of the cases of facial paralysis of the idiopathic—although not stated? The prognosis in all reported cases has been excellent. This patient had also auditory nerve symptoms. He had slight dizziness, which persisted. The treatment consisted of galvanism and tonic treatment. The patient was recovering function and reacting very nicely to the treatment.

The next case was one of a young lady, who was referred to the speaker three weeks before by a doctor in Rochester, New York, with a history of having had a great number of nasal operations performed—the patient said twenty-five—for headache and profuse nasal discharge. This discharge is of a mucous character, with considerable fibrin pus. Culture shows pneumococci. In spite of treatment, it continued. The an-

trum was punctured, washed out, and a cast came through the antral opening, which was perfectly round in shape. There was no pus—just a fibrinous exudate. The principal symptom is intense headache. There is a drawing back of the head. Vaccines have not helped at all, and, in fact, no treatment seems to be of avail. Consultation with neurologists confirmed Dr. Beck in his opinion, but he would not say anything definite about that until he had had Dr. Grinker's opinion in discussion regarding the diagnosis. Personally, he could say nothing about the etiology of the case. The patient is unable to breathe through the nose, and cannot blow the nose—still there is a space between the palate and the posterior wall of the pharynx, and the nares are patent.

DR. O. J. STEIN inquired whether the discharge continued while the patient was asleep, to which Dr. Beck replied that it was less during sleep. Bromids did not seem to affect the headache at all. The X-ray picture of her sinuses showed them to be affected, either postoperative or by the process.

The next patient was one of typical Froehlich's syndrome of hypopituitarism, enlargement of the anterior lobe of the hypophysis. The patient had the cardinal symptoms of loss of the hair on the body, and excessive fat over the body. Sexual function was lost. There was complete blindness of the right eye, and half blindness or lateral hemianopsia in the left eye. There was no headache, and no pain. The case had been gone over thoroughly from every point, and the diagnosis was not difficult. The reason for showing the patient was to speak of the operation, which had been described by him on previous occasions, and performed quite a number of times on the cadaver. By this method the hypophysis was reached through the antrum, following the Jansen method of opening the sphenoid by way of the antrum, through the posterior ethmoid and sphenoid, which is very easy in the normal case, where infection is not present. The sella then can be reached very easily. (Demonstration of two heads in which this operation had been performed.) The patient shown was operated upon three weeks ago under scopolamin-morphin anesthesia. He was given a few whiffs of ether when chiseling into the bone. The operation was perfectly easy, and its principal advantage was the fact that the operator could see

the structures he was going through. (Dr. Beck then showed illustrations of the different steps of the operation.) The X-ray in this case showed a very large sella turcica, and the stereoscopic picture was particularly valuable. The patient experienced very severe pain after the operation, of a neuralgic character, which necessitated keeping him under opiates. The enlargement of the gland was due to a cyst.

The point for consideration was: It was a cyst—will it recur? If so, there is a possibility of it causing pressure symptoms. The reason for operation was to conserve the little vision remaining.

DR. ROBERTSON asked if there was any pus present?

DR. BECK answered that there was none—the case was normal in that respect.

The next patient, a young girl, was one of a family of otosclerotics. Her principal complaint was severe pain in the front of the head and along the neck, with gradual development of symptoms of left-sided paralysis of the larynx, tongue, palate and shoulder—that is, of the ninth, tenth, eleventh and twelfth nerves. Besides that, one portion of the trifacial was also affected. She gave a history of having had tubercular glands when very young. The X-ray showed a calcareous gland in the neck. There is also a spontaneous nystagmus to both sides, slightly rotary. The case was diagnosed by one neurologist as a multiple sclerosis. The speaker made a diagnosis of pressure on these nerves from this latent tuberculosis, which diagnosis was concurred in by another neurologist. Canfield, who saw the case, said it was an intracranial proposition. Operation was performed by the speaker, when he dissected the nerves, and surrounded them with Cargile membrane. She is recovering the function of the tongue and the atrophy is disappearing. The larynx is beginning to move. The pains have disappeared; she is able to move the shoulder again, and the palate is also markedly improving. It was a case of pressure on the nerves by an old calcareous tuberculosis, lit up by a fresh attack. Reaction to tuberculin was absolutely negative a number of times.

DISCUSSION.

DR. JULIUS GRINKER, by invitation, said, with reference to the case of herpes zoster, that the explanation of its causation by a peritonsillar abscess because it began in the same way as the peritonsillar abscess appeared to him somewhat far-fetched. Since Hunt's masterly description of this type of zoster we know of its existence as one kind of zoster, but not as having a specific etiology. To say that a patient develops an otitic zoster, although entitled to a peritonsillar abscess, would be equivalent to a belief in some sort of compensatory function of zoster—which is improbable.

Dr. Beck's second case, the girl with the profuse serous discharge from the nose, he believed to be one of hysteria. Instead of the mucous colitis of hysteria and neurasthenia—a well-known phenomenon—we have the unusual symptom of hysterical rhinorrhea.

Referring to the case with multiple nerve lesions, the relief obtained by Dr. Beck's operation of removal of glands in the immediate vicinity of the nerves, proves his diagnosis of pressure neuritis, and admonishes one always to search for some tangible and removable cause in peripheral neuritis, especially if confined to one side.

The case of hypophyseal disease of the Froehlich type was interesting from the diagnostic viewpoint, but more so because Dr. Beck had discovered a new route to the hypophyseal region. Though easily reached by Dr. Beck—being at home in those parts—he thought there was quite a distance to travel. This route in certain cases seemed to him preferable to the one followed by McArthur and Frazier, whose operation by comparison must be considered formidable and not devoid of the usual dangers accompanying cranial operations.

DR. R. H. BROWN, regarding the second case, asked as to whether, in examining the nose, there was any marked turgescence present: These hysteric cases remind one very much of cases of diabetes insipidus, where there is a perfect flood of blood to a certain part (which did not show locally on examination), and he was interested in knowing whether, in a part like the nose, which could be examined, this local congestion showed.

DR. H. W. LOEB, of St. Louis, spoke about the case of

paralysis, which he had had the opportunity to see before, during and after operation. Dr. Beck said that she had a nystagmus to both sides, but he had a distinct recollection that the nystagmus was to the opposite side. It was on that basis that Dr. Canfield thought that the case was intracranial, and for that reason the speaker agreed with him. Dr. Beck had insisted on the condition being one of pressure in that region, and the speaker simply rose to state that Dr. Beck's view was correct, and the rest of the men who saw the case were wrong.

DR. CHARLES M. ROBERTSON referred to the hyperpituitary case. If this had been a tumor of the hypophysis, the young man probably would not have had the happy result seen at the present time. Dr. Robertson had reported a case to the society two or three years ago—when Dr. Pierce had a case—upon whom he operated by this route. It is a very simple operation, and the only thing to contend with is the sphenomaxillary artery. Dr. Robertson's was a cyst case, and recurred after the pituitary body was relieved of the pressure, but the cyst came down into the nose and was removed with the dura until the healthy dura was found by section afterwards. The man is still well, showing that these cases do get well of cysts under the pituitary body. Cases of tumor he would pass on to the surgeon, but in cysts he thinks that they belong to the field of the laryngologist and otologist, and this method is the shortest, safest and easiest route to the pituitary.

DR. BECK, in closing the discussion, wanted to take issue with Dr. Grinker. We do not have to have an abscess in order to have an infection.

Regarding the little girl, he showed her in order to have some of the members and guests express their opinions as to the question of hysteria. That was his diagnosis. The similarity between a mucous colitis or an excessive quantity of urine in a hysterical patient and the condition present in the young girl shown was the very point he had wished to have brought out.

Answering Dr. Brown's question, there was no turgescence present in this case. This brought him to a point made by Dr. Grinker as to the posterior lobe. He believes it is considered that diabetes insipidus is the symptom present when the posterior and middle lobes are involved, and that the anterior lobe involvement only produces the Froehlich syndrome.

Dr. GRINKER said that involvement of the anterior lobe produces acromegaly.

Dr. BECK said Dr. Loeb was wrong in the point of unilateral nystagmus. The patient now has bilateral nystagmus. Canfield said it was intracranial because it was slightly rotary.

Regarding the young man, it was, of course, a case of either tumor or cyst—it made no difference. He would rather it had been a tumor, because a cyst has more likelihood of recurring than a tumor.

INDEX OF AUTHORS.

- ABBOTT, W. J., 83.
 Abraham, J. H., 919.
 Adair-Dighton, 81.
 Adler, L., 189.
 Albanus, 255.
 Albrecht, W., 268.
 Alexander, L. D., 97.
 Ard, 452.
 Arnoldson, 262.
 Auerbach, J., 257.

 BABBITT, JAMES A., 607, 714,
 717, 719.
 Ballin, M. J., 224.
 Bar, 264.
 Barany, 253.
 Barnhill, John F., 381, 641, 644,
 647, 669, 686.
 Beck, Jos. C., 166, 207, 215, 488,
 498, 732, 736, 744, 747, 749, 790,
 887, 902, 910, 922, 939, 944, 954,
 957, 959.
 Beck, O., 156.
 Berens, T. Passmore, 469, 477,
 478, 483, 691, 726, 930, 933.
 Billings, W. C., 259.
 Birkett, H. S., 632.
 Blackwell, H. B., 588, 702, 705,
 706.
 Boot, G. W., 509, 518, 945, 948.
 Booth, D. S., 206.
 Borden, C. R. C., 222, 906.
 Bowers, W. C., 454, 455.
 Bowles, 708.
 Bradburne, S. A., 239.
 Braislín, W. C., 198, 402, 473, 932.
 Braun, A., 9, 368, 440, 441, 456,
 463, 701.
 Briggs, H. H., 529, 922.
 Brophy, T. W., 490, 503.
 Brown, H. Beattie, 600, 704.
 Brown, J. E., 874.
 Brown, R. H., 734, 939, 958.
 Bryan, Jos. H., 627, 686.
 Bryant, W. S., 190, 446, 897, 931.

 CALDERA, 262.
 Canesteo, C., 148.
 Canfield, R. B., 93.
 Carter, W. W., 779.

 Casselberry, Wm. E., 631, 639,
 657, 666, 678, 680, 682.
 Cavanaugh, J. A., 503.
 Chamberlin, W. B., 896, 911.
 Chambers, T. R., 164, 193, 913,
 917, 920.
 Citelli, 250.
 Clark, J. Payson, 630, 638, 683.
 Coakley, C. G., 646, 652, 653, 661,
 669, 670, 671, 697.
 Coates, G. M., 232, 912.
 Cocks, G. H., 200.
 Coffin, Lewis A., 660, 670, 695.
 Cohen, L., 924.
 Cohn, Felix, 189, 192, 431, 436.
 Corwin, Arthur M., 733, 734, 735.
 Corwin, T., 921.
 Cott, G. F., 887, 921.
 Craig, R. H., 445, 887.
 Crowe, S. J., 225.
 Curtis, H., 451.
 Curtis, H. H., 912, 915.

 DABNEY, VIRGINIUS, 668.
 Damm, 256.
 Danziger, E., 406, 436, 439, 442,
 446, 456.
 Davis, D. J., 514, 518.
 Davis, George E., 460, 597, 713,
 714, 716.
 Day, E. W., 230, 896, 902, 903.
 Dean, L. W., 509, 727, 729, 730,
 748, 749.
 De Carli, 216.
 Delavan, D. Bryson, 634, 642, 656,
 660, 684, 691.
 Dench, E. B., 237, 465, 473, 474,
 722, 725, 934, 935, 936.
 Dixon, 702, 708.
 Dougherty, 452.
 Duel, A. B., 193, 197, 447, 450,
 464, 467, 468, 472, 718, 893, 905,
 933, 934.
 Dworetzky, Jullus, 835.
 Dwyer, James S., 704.

 EAGLETON, W. P., 234, 432, 715,
 716, 936.
 Emerson, F. P., 226, 901, 913.
 Emerson, Linn, 700, 712, 713.

- FLETCHER, J. R., 489, 501, 743, 747, 749, 940, 942.
 Fowler, E. P., 233, 904, 905.
 Fraser, J. T., 220.
 Frazier, C. H., 270.
 Freer, O., 496.
 Freystadt, 263.
 Friedberg, S. A., 123, 214, 215, 293, 519, 521, 522, 908, 913.
 Friesner, L., 9, 410, 439, 440, 441, 456, 461, 713, 717.
 Fruewald, 269.
 GOLDMANN, R., 260.
 Gomperz, 254.
 Goodale, J. L., 273, 640, 635, 682.
 Grayson, C. P., 914, 917.
 Grinker, J., 944, 957, 959.
 Gruening, E., 464, 467, 468, 480, 483, 484, 715, 719, 724, 726, 933.
 Guggenheim, L. K., 208, 214.
 Guisex, 269.
 Guttman, J., 430.
 Gyergyal, A. V., 242.
 HALSTED, T. H., 261, 677, 693.
 Harris, T. J., 477, 479, 481, 482, 483, 722, 892, 903, 913, 931.
 Haseltine, B., 739.
 Haskin, W. H., 450, 453, 465, 467, 471, 477, 480, 582, 704, 707, 709, 720, 724, 931, 933, 935.
 Hayes, H., 443, 448, 706.
 Heller, 200.
 Henke, F., 252, 259.
 Henninger, 236.
 Holinger, J., 489, 502, 508, 739, 741, 745, 943, 944.
 Holmes, E. M., 138, 194, 202, 286, 555.
 Hopkins, F. E., 683, 685, 871, 878.
 Horn, John, 765.
 Hubbard, Thos., 625, 648, 660, 664, 679.
 Hudson-Makuen, G., 244, 887.
 Hurd, L. M., 920.
 IGLAUER, S., 235, 886, 891, 903.
 Ingals, E. Fletcher, 373, 519, 521, 522, 645, 657, 660.
 Ingersoll, J. M., 404, 633, 635.
 JACKSON, CHEVALIER, 659, 882, 887.
 Jacobs, L., 257.
 Jarecky, 193, 199.
 Johnston, R. H., 266.
 KAHN, H., 184, 208, 211, 213, 941, 944.
 Kelper, G. F., 53.
 Kenefick, J. A., 481, 721, 722, 723.
 Kenyon, E. L., 486, 489, 938, 949, 951, 954.
 Kerrison, P. D., 455, 459, 474, 930, 933, 935.
 Killian, G., 263.
 Kirkendall, J. T., 902, 911.
 Kopetzky, S. J., 391, 428, 431, 898.
 Kotz, R., 241.
 Kubo, 255, 256.
 LAKE, R., 239.
 Lederman, M. D., 201, 395, 432, 435, 440, 707, 708.
 Leidler, 248.
 Levinstein, 258.
 Levy, R., 887, 890, 892.
 Lewinsohn, R., 78.
 Lewis, Robert, 464, 466, 473, 723, 724.
 Lewy, A., 233, 592, 735, 740.
 Lockard, L. B., 269, 323, 698.
 Loeb, H. W., 209, 638, 668, 685, 687, 859, 958.
 Long, C. H., 939.
 Love, J. K., 240.
 Luders, C., 241.
 Lutz, S. H., 377, 464, 467, 478, 481, 483, 721, 930, 954.
 Lynch, R. C., 59, 917.
 MACLAY, O. H., 489, 502, 518, 939, 954.
 Marschik, 271.
 Masland, H. C., 257.
 Matte, 252.
 Maurice, 249.
 Mayer, Emil, 629, 630, 632, 655, 660, 678, 691.
 McBean, G. M., 419, 509, 514.
 McCowen, M., 485.
 McCullagh, 713, 714.
 McKenty, John E., 672.
 McKernon, J. F., 469, 471, 724, 934, 935, 936.
 Medling, 718.
 Mefford, W. T., 746.
 Milligan, W., 218, 220, 238.
 Mosher, H. P., 17, 658, 662, 670, 881, 887.
 Mulholland, 452.
 Myles, R. C., 194, 667, 705.

- OPDYKE, 434.
 Opie, E. L., 210.
 Oppenheimer, S., 193, 202, 433,
 448, 896, 904.
 Orloff, 245.
- PAGE, J. R., 161, 193, 474, 720,
 935.
 Perkins, C. E., 413, 431, 438, 441,
 440, 451, 784.
 Peter, L. C., 759.
 Pfeiffer, 267.
 Phillips, W. C., 224, 432, 436, 467,
 471, 475, 476, 726, 896, 902, 931,
 933, 935.
 Pierce, N. H., 913.
 Plummer, E. M., 17.
 Pynchon, E., 734.
- QUINLAN, 705.
- RANDALL, B. ALEXANDER, 648,
 652, 687.
 Reik, H. O., 909.
 Rethi, L., 265.
 Rhein, J. H. W., 244.
 Richards, G. L., 921.
 Richards, J. D., 456, 483.
 Richardson, Chas. W., 628, 629,
 676, 689, 894.
 Richardson, J. J., 335.
 Robertson, C. M., 492, 496, 510,
 948, 953, 957, 958.
 Robinson, 707.
 Roe, J. O., 916, 926.
 Ross, T. W. E., 216.
 Roy, Dunbar, 912.
 Ruttin, E., 237.
- SAUER, W. E., 215.
 Schmiegelow, E., 523.
 Scholz, R. P., 212, 214.
 Segura, 268.
 Shambaugh, G. E., 111, 204, 206,
 228, 509, 507, 509, 515, 640, 675,
 737, 741, 745, 937, 940, 948.
 Sharp, J. C., 74.
 Sheppard, J. E., 473, 474, 481, 482,
 483, 574, 864, 892, 897.
- Shurly, Burt R., 637, 647, 672,
 673, 676, 677.
 Skillern, G. Ross, 668, 884, 915.
 Sluder, Greenfield, 211, 650, 669,
 673, 692, 755.
 Smith, Harmon, 264, 650, 692.
 Smith, S. MacCuen, 896.
 Smyth, 432.
 Solis-Cohen, J., 643.
 Sonnenschein, R., 235, 619, 746,
 748, 954.
 Spencer, F. R., 916, 924.
 Steel, Geo. E., 416, 443.
 Stein, Otto J., 116, 207, 210, 212,
 518, 520, 521, 727, 743, 746, 749,
 939, 956.
 Strauss, 428.
 Swain, Henry L., 444, 628, 631,
 639, 643, 648, 659, 675, 679, 684,
 697.
- THEIMER, K., 217.
 Theisen, C. F., 1, 569.
 Thompson, J. A., 896, 918, 921.
 Thompson, J. J., 578.
 Thomson, St. Clair, 688.
 Thost, A., 267.
 Toeplitz, M., 934.
 Torrey, G., 485, 490.
 Tymeson, 700, 713.
- VAIL, 714.
 Verel, R., 217.
 Voislavsky, A. P., 455, 700, 701,
 707, 712, 911.
- WALES, E., 563.
 Well, A. I., 928.
 Welty, C. F., 66, 227.
 Whiting, Fred, 720, 930, 933.
 Wilson, 202, 704, 708.
 Wilson, J. G., 205, 209, 259.
 Wittmaack, 242.
 Wood, Geo. B., 661, 671, 674.
- YANKAUER, S., 194, 885, 891.
 Yates, D. G., 228.
 Young, H. B., 827.
- ZOGRAFFDES, A., 241.

INDEX OF TITLES.

- ABSCCESS**, alveolus, Berens, 477.
Abscess, brain, Dench, 722; Fruewald, 269; Henke, 252; Ruttin, 237.
Abscess, brain, instrument for exploring, Page, 720.
Abscess, cerebellum, Adair-Dighton, 81; Perkins, 413, 441.
Abscess, cervical, of aural origin, De Carlh, 216.
Abscess, retropharynx, Billings and Wilson, 259.
Abscess, subperiosteal, with mastoiditis, Haskin, 931.
Abscess, temporosphenoidal, Dean, 726; Duell, 469; Emerson, 226, 713; Ingersoll, 404.
Acoustic nerve, curable affections, Lewy, 233.
Acoustic nerve, effects of salvarsan, Toeplitz, 934.
Actinomyces-like granules in the tonsils, Davis, 514.
Adenocarcinoma nose, Alexander, 97.
Adenoids, Lewis, 464.
Air we breathe, Hubbard, 624.
Alae nasi, collapse, Damm, 256.
Alveolus abscess with fragment of tooth in situ, Berens, 477.
Anaphylaxis in horse asthma, Goodale, 273, 635.
Anesthesia, local, in mastoid operation, Boot, 945.
Angina, Vincent's, Richardson, 335.
Appendicitis with ear pain, Kerrison, 935.
Asthma, bronchoscopic treatment, Keiper, 53.
Asthma, horse, Goodale, 273, 635.
Asthma, nasal relations, Abbott, 83.
Auditory affections and ocular imbalance, Bradburne, 239.
Auditory disturbances, relief, Eagleton, 234.
Aural complications of exanthemata, Borden, 906; Friedberg, 908.
Aural douching followed by suppuration, Harris, 482.
Aural infection with streptococcus capsulatus, Perkins, 784.
Aural vertigo, Lake, 239.
BACILLUS fusiformis causing meningitis and brain abscess, Fruewald, 269.
Bacteremia, Sheppard, 864, 892.
Bacteriology chronic ear, Dwyer, 704.
Blindness cured by operation on ethmoid, Loeb, 685, 859.
Bone transplantation for postoperative mastoid wound, Ballin, 224.
Brain abscess, Dench, 722; Fruewald, 269; Hencke, 252; Ruttin, 237.
Brain abscess, instrument for exploring, Page, 720.
Brain cyst, Kahn, 941.
Bronchoscopic treatment of asthma, Keiper, 53.
Bronchoscopy, fluoroscopic, Ingals and Friedberg, 519.
Bronchoscopy limitations, Jackson, 655.
Bronchus surgery, experimental, Caldera, 262.
Bulb abnormality, Harris, 479.
Bulbar paralysis, Freystadt, 263.
CANCER esophagus, Guisez, 269.
Cancer larynx, Schmiegelow, 523; Thomson, 688.
Cancer larynx, radium therapy, Beck, 166.
Cauterization nasal mucosa, Wales, 563.
Cavernous sinus, relation to sphenoid sinus, Sluder, 755.
Cavernous sinus thrombosis, Braun, 368, 440; Friesner, 410, 440.
Cerebellum abscess, Adair-Dighton, 81; Perkins, 413-441.
Cerebral symptoms complicating latent mastoiditis, Kopetzky, 391, 428.

- Cervical abscess of aural origin, De Carl, 216.
 Cholesteatoma, Phillips, 475.
 Cleft palate speech aspects, Kenyon, 949.
 Contrecoup fracture skull, Chambers, 164, 193.
 Cord nodule, Shambaugh, 937.
 Cyst brain, Kahn, 941.
- DEAF reeducation, Maurice, 249.
 Deafness middle ear, Shambaugh, 228.
 Deafness prevention, Hudson-Makuen, 244.
 Deafness, sociologic aspect, Young, 827.
 Deafness, sporadic congenital, Love, 240.
 Deafness, syphilitic, Love, 240.
 Deafness, total, McKernon, 934.
 Decompression in intracranial complications of otitic origin, Milligan, 238.
 Dehiscence tympanic roof, Lewis, 723.
 Dementia precox, improvement after double radical mastoidectomy, Davis, 713.
 Digestive tract, mycosis, Segura, 268.
 Diphtheria and middle ear diseases, Borden, 222.
 Diphtheria carriers, vaccines in, Weil, 928.
 Diphtheria ear, Haskin, 450.
 Drum discoloration, Sheppard, 480.
 Drum, relaxed, Hayes, 443.
 Dura mater sarcoma, Barnhill, 381.
- EAR, chronic, bacteriology, Dwyer, 704.
 Ear complications of typhoid fever, Holmes, 555.
 Ear diphtheria, Haskin, 450.
 Ear diseases treated by heat, Maljutin, 245.
 Ear furunculosis, Brown, 600, 704.
 Ear infection treated with vaccines, Haskin, 582, 704.
 Ear pain with appendicitis, Kerrison, 935.
 Ear syphilis, Stein, 116, 207.
- Empyema accessory sinuses, Coakley, 661; Coffin, 660; Wood, 661.
 Empyema maxillary sinus, Masland, 257.
 Endocranial complications, Braun and Friesner, 456.
 Endocranial complications of otitic origin, Milligan, 238.
 Endocranial complications of suppurative labyrinthitis, Braun and Friesner, 9.
 Endolaryngeal operation for cancer of the larynx, Thomson, 688.
 Endonasal operation for hypophysis tumor, Halstead, 693.
 Endoscopy, Johnston, 266.
 Environmental surgery in otology, Barnhill, 641.
 Epiglottitis infection, Theisen, 569.
 Epipharynx in childhood, Holmes, 138, 194.
 Equilibrium and semicircular canals, Shambaugh, 111, 204.
 Erysipelas following mastoid operation, Fraser, 220.
 Esophagoscope, Lewinsohn, 78.
 Esophagoscopy, Friedberg, 122, 214.
 Esophagus anatomy, Mosher, 881.
 Esophagus anomalies, Mosher, 881.
 Esophagus cancer, Guisez, 269.
 Esophagus stenosis, Mosher, 881.
 Esophagus tuberculosis, Lockard, 269.
 Ethmoid disease causing blindness and scintillating scotoma, Loeb, 685, 859.
 Ethmoiditis with external fistula, Shambaugh, 937.
 Eustachian tube dilatation, Gyergya, 242.
 Eye affections influenced by nose, Loeb, 859.
 Eye symptoms in chronic sinus disease, Peter, 759.
- FACIAL paralysis, Emerson, 700.
 Facial paralysis with herpes zoster oticus, Beck, 954.
 Fever significance in mastoiditis, Verel, 217.
 Fibromyxoma nasopharynx, Brown, 874.

- Fifth nerve relation to sphenoid sinus, Sluder, 755.
- Fistula, external, with frontal sinusitis and ethmoiditis, Shambaugh, 937.
- Fistula parotid following mastoid operations, Canestoe, 148.
- Fistula semicircular canal, Berrens, 478.
- Fistula symptom in nonsuppurative ear diseases, Beck, 156.
- Fluoroscopic bronchoscopy, Ingals and Friedberg, 519.
- Forceps ridge, Lutz, 464.
- Fourth nerve, relation to sphenoid sinus, Sluder, 755.
- Fracture bony meatus, Kerrison, 474.
- Fracture skull, contrecoup, Chambers, 164, 193.
- Fracture skull followed by meningitis, Eagleton, 936.
- Frontal sinus disease, Holinger, 741.
- Frontal sinusitis with external fistula, Shambaugh, 937.
- Furunculosis ear, Brown, 600, 704.
- GALVANOCAUTERY** in larynx tuberculosis, Bar, 264.
- Gout upper air passages, Thost, 267.
- Growths larynx, removal, Lynch, 917.
- HALLUCINATIONS** in otitis media suppurativa chronica, Kenefick, 721.
- Hallucinations of hearing, Rhein, 244.
- Hearing improvement after radical mastoid operation, Bowers, 454.
- Heat treatment of diseases of the ear, Maljutin, 245.
- Heath operation, Plummer and Mosher, 17.
- Hemophilia in male child, Sheppard, 480.
- Herpes zoster oticus with facial paralysis, Beck, 954.
- Hiss leucocyte extract in mastoiditis caused by streptococcus capsulatus mucosus, Has-kin, 471.
- Horse asthma, Goodale, 635.
- Hot air in laryngology, Albrecht, 268.
- Hydrorrhea nasal, Kahn, 184, 211.
- Hypophysis tumor, Halstead, 693.
- INFECTION**, aural, with streptococcus capsulatus, Perkins, 784.
- Influenza, chronic, Lockard, 323, 698.
- Injury palate with stammering, Kenyon, 951.
- Intracranial nerve division, Frazier, 270.
- Intranasal operation for nasolacrimal stenosis, Holmes, 286.
- JARISCH - HERXHEIMER** reaction on hearing after treatment by salvarsan, Thelmer, 217.
- Jugular bulb injury following myringotomy, Page, 164, 193.
- Jugular resection, Lederman, 395, 432.
- LABYRINTH** inflammation, Henninger, 236.
- Labyrinth operation, Dench, 237; Leidler, 248; Matte, 252; Welty, 66.
- Labyrinth studies, Babbitt, 714.
- Labyrinth vertigo, Matte, 252; Milligan, 220.
- Labyrinthitis, acute, Emerson, 700.
- Labyrinthitis following mumps, Phillips, 935.
- Labyrinthitis, serous diffuse, complicating acute purulent otitis media, Danziger, 406, 436.
- Labyrinthitis, suppurative, complicated by cavernous sinus thrombosis, Braun, 368, 440.
- Labyrinthitis, suppurative, endocranial complications, Braun and Friesner, 9.
- Laryngitis submucosa subglottica, Richardson, 676.
- Laryngocele ventricularis, Shambaugh, 644.
- Laryngofissure, Schmiegelow, 523.
- Laryngopharynx lymphoma, Casselberry, 680.
- Laryngoscopy, direct, Smith, 264.
- Laryngoscopy, suspension, Killian, 263; Levy, 890.

- Larynx cancer, Schmiegelow, 523; Thomson, 688.
- Larynx cancer, radium therapy, Beck, 166.
- Larynx growths, intrinsic, Lynch, 917.
- Larynx, hot air treatment, Albrecht, 266.
- Larynx infection, Theisen, 569.
- Larynx lupus, Mayer, 630.
- Larynx papilloma, Hopkins, 683, 878.
- Larynx paralysis in acute bulbar paralysis, Freystadt, 263.
- Larynx polypus with tetany, Canfield, 93.
- Larynx radiography, Rethi, 265.
- Larynx safety pin, Smith, 264.
- Larynx tuberculosis, Arnoldson, 262; Bar, 264; Dworetzky, 835.
- Lateral sinus operation, Lederman, 395, 432.
- Lateral sinus thrombosis, McKernon, 469.
- Leprosy upper respiratory tract, Horn, 765.
- Lip reading, Torrey, 485.
- Lupus larynx, Mayer, 630.
- Lupus nose, Albanus, 255.
- Lymphoma laryngopharynx, Caselberry, 680.
- MASTOID cavity obliteration, Iglaue, 235.
- Mastoid opened for pain, Lutz, 930.
- Mastoid operation followed in twelve hours by cavernous sinus thrombosis, Friesner, 410, 440.
- Mastoid operation followed by erysipelas, Fraser, 220.
- Mastoid operation followed by parotid fistulae, Canestee, 148.
- Mastoid operation, radical, Emerson, 700, 713; Milligan, 238; Sharp, 74; Welty, 227; Yates, 228.
- Mastoid operation, radical, dressing with scarlet red, Ross, 216.
- Mastoid operation, radical, in dementia precox, Davis, 713.
- Mastoid operation under local anesthesia, Boot, 945.
- Mastoid wound, postoperative, and tibial bone transplantation, Ballin, 224.
- Mastoiditis, acute, Emerson, 713.
- Mastoiditis due to streptococcus capsulatus mucosus, Gruening, 464.
- Mastoiditis from streptococcus capsulatus mucosus treated with Hiss leucocyte extract, Haskin, 471.
- Mastoiditis, latent, complicated by cerebral symptoms, Kopetzky, 391, 428.
- Mastoiditis, resolving, Blackwell, 588, 702.
- Mastoiditis, significance of fever, Verel, 217.
- Mastoiditis with subperiosteal abscess, Haskin, 931.
- Maxillary empyema, Masland, 257.
- Maxillary sinus, Levinstein, 258.
- Maxillary sinus disease, Brophy, 490.
- Maxillary sinus, vacuum disease, Lynch, 59.
- Maxillary sinusitis, chronic, Kubo, 255.
- Meatus, bony fracture, Kerrison, 474.
- Membrana tympani regeneration, Coates, 232.
- Meningitis cerebrospinal, Bryant, 897.
- Meningitis chronic, Thomson, 578.
- Meningitis following fracture of skull, Eagleton, 936.
- Meningitis from bacillus fusiformis, Fruewald, 269.
- Meningitis, internal acute, Rutlin, 237.
- Meningitis occurring late in mastoidectomy convalescence, Steel, 416, 443.
- Meningitis of otitic origin, Milligan, 218.
- Meningitis, operative treatment, Kopetzky, 898.
- Meningitis, otitic, Day, 230; Kotz, 241.
- Middle ear conditions and disturbed metabolism, Sheppard, 574.
- Middle ear deafness, obstructive, Shambaugh, 228.

- Middle ear diseases in diphtheria and scarlet fever, Borden, 222.
 Middle ear syphilis, Luders, 241.
 Middle ear tuberculosis, Briggs, 529, 922.
 Mumps followed by labyrinthitis, Phillips, 935.
 Mycosis upper air tract and digestive tract, Segura, 268.
 Myringotomy followed by injury to jugular bulb, Page, 161, 193.
 Myxosarcoma nasopharynx, Ingals, 373, 649.
 NASAL accessory sinuses, Davis, 753.
 Nasal development, dynamics in resection of the septum, Carter, 779.
 Nasal hydrorrhea, Kahn, 184, 211.
 Nasal mucosa cauterization, Wales, 563.
 Nasal septum resection, Carter, 779.
 Nasolacrimal stenosis, Holmes, 286.
 Nasopharynx, fibromyxoma, Brown, 874.
 Nasopharynx myxosarcoma, Ingals, 373, 649.
 Necrosis superior maxilla, Dean, 726.
 Neosalvarsan, Pfeiffer, 267.
 Nerve division, intracranial, Frazier, 270.
 Nodule cord, Shambaugh, 937.
 Nose, adenocarcinoma, Alexander, 97.
 Nose, influence on eye affections, Loeb, 859.
 Nose, influenza, chronic, Lockard, 323.
 Nose, pathology, Beck, 790.
 Nose, relations of bronchial asthma, Abbott, 83.
 Nystagmus, Fowler, 904.
 OCULAR imbalance and auditory affections, Bradburne, 239.
 Operative surgery of the nose, throat and ear, Loeb, 750.
 Otitis and concomitant association with other diseases, Cohn, 189.
 Otitis externa furunculosa, Zografides, 241.
 Otitis media catarrhalis chronica, Lutz, 377.
 Otitis media suppurativa acuta complicated by diffuse serous labyrinthitis, Danziger, 406, 436.
 Otitis media suppurativa chronica causing temporosphenoidal abscess, Ingersoll, 404.
 Otitis media suppurativa chronica with cholesteatoma, Phillips, 475.
 Otitis media suppurativa chronica with hallucinations, Kenefick, 721.
 Otitis media suppurativa with dehiscence of tympanic roof, Lewis, 723.
 Otolaryngology, environmental surgery, Barnhill, 641.
 Otorrhea, persistent, in infants and young children, Phillips, 224.
 Otosclerosis, Citelli, 250.
 Otosclerosis treated by Roentgen ray, Ortloff, 245.
 Ozena, Auerbach, 257; Jacobs, 257.
 PAIN in ear with appendicitis, Kerrison, 935.
 Palate cleft, speech aspects, Kenyon, 940.
 Palate injury with stammering, Kenyon, 951.
 Palate, soft, and pharynx, correlated action, Sluder, 651.
 Papilloma larynx, Hopkins, 683, 878.
 Paraffin in ozena, Auerbach, 257.
 Paralysis, bulbar, Freystadt, 263.
 Paralysis, facial, Emerson, 700.
 Paralysis, facial, with herpes zoster oticus, Beck, 954.
 Paralysis larynx, Freystadt, 263.
 Paranasal sinus suppuration, Sluder, 650.
 Parotid fistulae following mastoid operations, Canestee, 148.
 Parotid glands, septic infection, Hopkins, 683, 871.
 Pathology, histologic, of the nose, Beck, 790.
 Pemphigus, Sonnenschein, 619, 747.
 Pharynx and soft palate, correlated action, Sluder, 651.

- Phlegmonous inflammation tonsil, Henke, 259.
- Pneumonia following radical mastoid operation, Emerson, 713.
- Polyp sphenoid sinus, Kubo, 256.
- Polypus larynx with tetany, Canfield, 93.
- Punch, Lutz, 464.
- RADIOGRAPHIC pictures of the jaw, Haskin, 720.
- Radiography larynx, Rethi, 265.
- Radium in esophagus cancer, Gulsez, 269.
- Radium in laryngorhinology, Marschik, 271.
- Radium in papilloma larynx, Hopkins, 683, 878.
- Radium therapy in larynx cancer, Beck, 166.
- Reeducation of the deaf, Maurice, 249.
- Resonators, Sonnenschein, 235.
- Resection, septum, nasal, Carter, 779.
- Respiratory tract, upper, leprosy, Horn, 765.
- Retropharynx abscess, Billings and Wilson, 259.
- Rhinitis, atrophic, Jacobs, 257.
- Rhinology, optimistic, Randall, 687.
- Rhinoplasty, corrective, Cohen, 924.
- Ridge forceps, Lutz, 464.
- Roentgen ray in otosclerosis, Orloff, 245.
- SAFETY pin in larynx, Smith, 264.
- Salvarsan, effect on acoustic nerve, Toeplitz, 934.
- Salvarsan treatment and Jarisch-Herxheimer reaction, Theimer, 217.
- Sarcoma dura mater, Barnhill, 381.
- Sarcoma trachea, Ingersoll, 633.
- Scarlet fever and middle ear disease, Borden, 222.
- Scarlet red dressing in radical mastoid operations, Ross, 216.
- Scotoma, scintillating, cured by operation on ethmoid, Loeb, 685, 859.
- Semicircular canals and function of equilibrium, Shambaugh, 111, 204.
- Semicircular canal fistula, Berens, 478.
- Septum, nasal, resection, Carter, 779.
- Serum treatment, local, for phlegmonous inflammation faucial tonsil, Henke, 259.
- Silver nitrate applied to mouth of eustachian tube for tinnitus, Bralslin, 402.
- Sinus, cavernous, relation to sphenoid sinus, Sluder, 755.
- Sinus, cavernous, thrombosis, Braun, 368, 440; Friesner, 410, 440.
- Sinus disease, chronic, value of eye symptoms in, Peter, 759.
- Sinus, frontal, disease, Hollinger, 741.
- Sinus, lateral, operation, Lederman, 395, 432.
- Sinus, lateral, thrombosis, McKernon, 469.
- Sinus, maxillary, Levinstein, 258.
- Sinus, maxillary, disease, Brophy, 490.
- Sinus, maxillary, vacuum disease, Lynch, 59.
- Sinuses, nasal, empyema, Coffin, 660; Coakley, 661; Wood, 661.
- Sinus, paranasal, suppuration, Sluder, 650.
- Sinus, sphenoid, Grayson, 914.
- Sinus, sphenoid, disease, McBean, 419, 509.
- Sinus, sphenoid, polyp, Kubo, 256.
- Sinus, sphenoid, relation to cavernous sinus and third, fourth, fifth, sixth and vidian nerves, Sluder, 755.
- Sinus, superior, longitudinal thrombosis, Boot, 445.
- Sinus thrombosis, Crowe, 225.
- Sinusitis, frontal, with external fistula, Shambaugh, 937.
- Sinusitis, maxillary, Kubo, 255.
- Sixth nerve relation to sphenoid sinus, Sluder, 755.
- Skiagraph in thymus gland enlargement, Delavan, 645.
- Skull fracture, contrecoup, Chambers, 164, 193.

- Skull fracture followed by meningitis, Eagleton, 936.
 Speech aspect of cleft palate, Kenyon, 949.
 Sphenoid sinus anatomy, Sluder, 755.
 Sphenoid sinus disease, McBean, 419, 509.
 Sphenoid sinus, exploratory opening, Grayson, 914.
 Sphenoid sinus polyp, Kubo, 256.
 Stammering with palate injury, Kenyon, 95.
 Stenosis esophagus, Mosher, 881.
 Stenosis, nasolacrimal, Holmes, 286.
 Streptococcus hemolyticus in lymphomata of laryngopharynx, Casselberry, 680.
 Streptococcus mucosus capsulatus and aural infection, Perkins, 784.
 Streptococcus mucosus capsulatus causing mastoiditis, Gruening, 464.
 Streptococcus mucosus capsulatus infection, Haskin, 471, 724.
 Subperiosteal abscess with mastoiditis, Haskin, 931.
 Superior maxilla necrosis, Dean, 727.
 Suppuration following aural douching, Harris, 482.
 Suspension laryngoscopy, Killian, 263; Levy, 890.
 Syphilis causing deafness, Love, 240.
 Syphilis of ear, Stein, 116, 207.
 Syphilis of middle ear, Luders, 241.
- TEMPOROSPHENOIDAL** abscess, Dean, 726; Duel, 469; Emerson, 713; Ingersoll, 404.
 Tetany with laryngeal polypi, Canfield, 93.
 Third nerve, relation to sphenoid sinus, Sluder, 755.
 Throat influenza, chronic, Lockard, 323.
 Thrombosis cavernous sinus, Braun, 368, 440.
 Thrombosis lateral sinus, Crowe, 225; McKernon, 469.
 Thrombosis superior longitudinal sinus, Boot, 945.
 Thymus gland enlargement, Delavan, 645.
 Thyroid disease, tonsil relation, Shurly, 673.
 Thyroid gland, perverted function, Shambaugh, 507.
 Thyroiditis, acute, complication or acute tonsillitis, Theisen, 1.
 Thyroiditis, suppurative, Theisen, 569.
- Tinnitus aurium, Bryan, 931; Fowler, 233; Wittmaack, 242.
 Tinnitus treated by nitrate of silver applied to the mouth of the eustachian tube, Brailsin, 402.
 Tonsil, faucial, phlegmonous inflammation, Henke, 259.
 Tonsil relation to thyroid diseases, Shurly, 673.
 Tonsil surgery, modern, Halsted, 261.
 Tonsil treatment, Goldmann, 260.
 Tonsil with actinomyces-like granules, Davis, 514.
 Tonsillectomy, Lewy, 592, 735.
 Tonsillitis, acute, complicated by acute thyroiditis, Theisen, 1.
 Tonsillotome, evolution, Friedberg, 293.
 Trachea perforation, Theisen, 569.
 Trachea, sarcoma, Ingersoll, 633.
 Tuberculosis esophagus, Lockard, 269.
 Tuberculosis larynx, Arnoldson, 262; Bar, 264; Dworetzky, 835.
 Tuberculosis middle ear, Briggs, 529, 922.
 Tympanic membrane perforation, cicatrization, Gomperz, 254.
 Tympanic roof dehiscence, Lewis, 723.
 Typhoid fever, ear complications, Holmes, 555.
- UPPER** air passages, diseases, Thompson, 918.
 Upper air passages, gout, Thost, 287.
 Upper air passages, mycosis, Segura, 268.
- VACCINATION**, autogenous, for recurrent lymphomata of the laryngopharynx, Casselberry, 680.
 Vaccines in chronic diphtheria carriers, Well, 928.
 Vaccines in ear infection, Haskin, 582, 704.
 Vaccines in furunculosis ear, Brown, 588, 704.
 Vacuum disease maxillary sinus, Lynch, 58.
 Vein anomaly, Phillips, 478.
 Vertigo, aural, Lake, 239.
 Vertigo, labyrinth, Matte, 252; Milligan, 220.
 Vestibular apparatus, relation to central nervous system, Barany, 253.
 Vidian nerves, relation to sphenoid sinus, Sluder, 755.
 Vincent's angina, Richardson, 335.

Annals of Otology, Rhinology and Laryngology

FOUNDED BY JAMES PLEASANT PARKER

H. W. LOEB, M. D., ST. LOUIS
EDITOR-IN-CHIEF.

COLLABORATORS

T. MELVILLE HARDIE, M. D., Chicago; JAMES T. CAMPBELL, M. D., Chicago; G. L. RICHARDS, M. D., Fall River; THOS. J. HARRIS, M. D., New York; J. L. GOODALE, M. D., Boston; SAMUEL E. ALLEN, M. D., Cincinnati; CLEMENT F. THEISEN, M. D., Albany; GEO. B. WOOD, M. D., Philadelphia; G. ALEXANDER, M. D., Vienna; WYATT WINGRAVE, M. D., London; A. JANSEN, M. D., Berlin; JOHN SENDZIAK, M. D., Warsaw; W. POSTHUMUS MEYJES, M. D., Amsterdam; N. R. H. BLEGVAD, M. D., Copenhagen; HENRY HORN, M. D., San Francisco; VICTOR F. LUCCHETTI, M. D., San Francisco.

PUBLISHED QUARTERLY
BY JONES H. PARKER,
MERMOD-JACCARD BUILDING,
ST. LOUIS, MO., U. S. A.

SUBSCRIPTION PRICE, \$4.00 PER ANNUM, IN ADVANCE.

Subscriptions in other countries of the Postal Union, 18 shillings.

ANNALS OF OPHTHALMOLOGY, \$4.00

Entered at the Postoffice at St. Louis, Mo., as Second-class Matter.

CONTENTS

LV.—Further Observation on Some Anatomic and Clinical Relations of the Sphenoid Sinus to the Cavernous Sinus and the Third, Fourth, Fifth, Sixth and Vidian Nerves. By Greenfield Sluder, M. D., St. Louis.....	755
LVI.—The Value of Eye Symptoms in the Diagnosis of Obscure Chronic Sinus Disease. By Luther C. Peter, A. M., M. D., Philadelphia.....	759
LVII.—Leprosy of the Upper Respiratory Tract With Report of a Case. By John Horn, M. D., New York.....	765
LVIII.—The Dynamics of Nasal Development—Its Bearing on Resection of the Septum. By William Wesley Carter, A. M., M. D., New York City.....	779
LIX.—Report of Cases of Aural Infection With the Streptococcus Capsulatus. By Charles E. Perkins, M. D., New York.....	784
LX.—Histologic Pathology of the Nose. By Joseph C. Beck, M. D., Chicago.....	790
LXI.—The Sociologic Aspect of Deafness, Congenital or Acquired in Early Life, With a Suggestion for a Betterment Through Indirect Effort. By H. B. Young, A. M., M. D., Burlington, Iowa.....	827
LXII.—Etiology and Prophylaxis of Tuberculous Laryngitis. By Julius Dworetzky, M. D., Otisville, New York.....	835
LXIII.—The Influence of the Nose on Eye Affections as Evidenced by a Case of Bilateral Blindness and One of Unilateral Scintillating Scotoma Cured by Operations on the Ethmoid Cells. By H. W. Loeb, M. D., St. Louis.....	859
LXIV.—The Clinical Significance of Bacteremia. By J. E. Sheppard, M. D., F. A. C. S., Brooklyn.....	864
LXV.—A Case of Septic Infection of the Parotid Glands Resulting Fatally. By F. E. Hopkins, M. D., Springfield.....	871
LXVI.—Two Cases of Extensive Fibromyxoma of the Nasopharynx. By John Edwin Brown, M. D., Columbus.....	874
LXVII.—A Case of Papilloma of the Larynx Treated by Radium. By F. E. Hopkins, M. D., Springfield.....	875

CONTENTS—Continued on Third Cover Page.

CONTENTS—Continued from Second Cover Page.

Society Proceedings881-960

Transactions of the American Laryngological, Rhinological and Otological Society.—Symposium; Stenosis of Esophagus; (a) Anatomy, Anomalies, Instruments and Technic; (b) Causes, Symptoms, Pathology, Diagnosis and Treatment—Suspension Laryngoscopy in Children—The Clinical Significance of Bacteremia—Treatment of Purulent Cerebrospinal Meningitis—The Operative Treatment of Meningitis; Supplementary Report and Analysis of Cases—The Observation of Nystagmus Through the Closed Eyelids—Symposium: Aural Complications of the Exanthemata; (a) Etiology, Diagnosis and Treatment; (b) The Etiology, Diagnosis and Treatment of the Aural Complications of the Exanthemata—The Exploratory Opening of the Sphenoid Sinus—A New Technic for the Removal of Intrinsic Growths of the Larynx—The Proper Fields of Medicine and Surgery in Diseases of the Upper Air Passages—Tuberculosis of the Middle Ear—Corrective Rhinoplasty—The Efficacy of Vaccines in the Treatment of Chronic Diphtheria Carriers.....881-929

New York Otological Society, Regular Meeting Tuesday, May 28, 1914.—Mastoid Opened for Relief of Pain—Pain, Tinnitus and Deafness With Sensation of Foreign Body for Four Years—Mastoiditis With Subperiosteal Abscess—The Effect of Salvarsan Upon the Acoustic Nerve—Supplementary Report in a Case of Total Deafness—Pain in the Ear and Over the Mastoid in Conjunction With Appendicitis—Labyrinthitis Following Mumps—Meningitis Following Fracture of Skull.....930-936

Chicago Laryngological and Otological Society, Regular Meeting, April 21, 1914.—Case of Singer's Nodule on the Vocal Cord—Case of Frontal Sinusitis and Ethmoiditis With an External Fistula—Brain Cyst—Case of Simple Mastoid Operation Under Local Anesthesia—Thrombosis of Superior Longitudinal Sinus—The Speech Aspects of a Case of Cleft Palate—Case of Injured Palate, With Stammering—Herpes Zoster Oticus, With Facial Paralysis.....937-960



